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CONTENTS OF PREVIOUS NUMBERS

OCTOBER, 1919. NUMBER 4.

The Caloric Requirements of Normal Infants and Children from Birth to Puberty. Fritz B. Talbot, M.D., Boston.

Influenzal Croup. Henry Lowndes Lynah, M.D., New York.

A Health Study of a Boy's School. Richard M. Smith, M.D., Boston.

Certain Nutritional Disorders of Children Associated with a Putrefactive Intestinal Flora. Langley Porter, M.D.; G. B. Morris, M.D., and K. F. Meyer, M.D., San Francisco.

Study of the Stools in Children's Institutions Showing the Incidence of Intestinal Parasitic Infections. L. R. DeBuys, B.S., M.D., New Orleans, and H. L. Dwyer, M.D., Kansas City, Mo.

Progress in Pediatrics: Review of Recent Literature on the New-Born. N. O. Pearce, M.D., Minneapolis.

Child Welfare. Report from International Congress.

Current Pediatric Literature.

NOVEMBER, 1919. NUMBER 5.

The Results of Thymus Extirpation in the Dog with a Review of the Experimental Literature on Thymus Extirpation. Edwards A. Park, M.D., and Roy D. McClure, M.D., Baltimore.

Current Pediatric Literature.

DECEMBER, 1919. NUMBER 6.

The Early Recognition of Hydrocephalus in Meningitis. Kenneth D. Blackfan, Baltimore.

Chemical Examination of the Blood in Children. A Study of One Hundred and Forty-Nine Cases. Henry Dwight Chapin, M.D., and Victor Carl Myers, Ph.D., New York.

The Rôle of the Antineuritic Vitamin in the Artificial Feeding of Infants. Amy L. Daniels, Ph.D., and Albert H. Byfield, M.D., with the Cooperation of Rosemary Loughlin, M.S., Iowa City, Iowa.

Stammering as a Disorder of Speech Dependent on Conditions of Child Development. Elmer L. Kenyon, M.D., Chicago.

Qualitative and Quantitative Changes in the Cerebrospinal Fluid of Various Diseases and Their Significance. A. Levinson, Chicago.

A Résumé of Some Experimental Studies on Cutaneous Hypersensitiveness. E. C. Fleischner, K. E. Meyer and E. B. Shaw, San Francisco.

Current Medical Literature.

Index to Volume 18.

JANUARY, 1920. NUMBER 1.

A Study of Achondroplasia. Introducing a New Symptom — Wedge-Shaped Vertebra. Thomas Foster Wheeldon, A.M., M.D., Richmond, Va.

The Food Value of the Milk of the Water Buffalo. William W. Cadbury, M.D., Canton, China.

Dextrose Tolerance in Atrophic Infants. P. M. Mattill, K. M. Mayer and L. W. Sauer, Chicago.

A Case of Hypersensitiveness to Cow's Milk. Edwards A. Park, M.D., Baltimore.

Clinical Department: A Case of Anomaly of the Diaphragm with Herniation into the Thorax of Certain Viscera Resulting in a Gastric and Intestinal Obstruction. L. R. DeBuys, B.S., M.D., New Orleans.

Progress in Pediatrics: Résumé on the Circulatory System. Review of Literature of 1917, 1918 and 1919. Henrietta Calhoun, M.B., Iowa City, Iowa.

FEBRUARY, 1920. NUMBER 2.

The Etiology of Arthritis Deformans in Children. Albert H. Byfield, M.D., Iowa City.

Calcium Metabolism of Infants and Young Children, and the Relation of Calcium to Fat Excretion in the Stools. L. Emmett Holt, M.D., Angelia M. Courtney and Helen L. Fales, New York.

Incidence of Protein Sensitization in the Normal Child. Horace Mitchell Baker, M.D., Boston.

Foreign Bodies in the Air and Food Passages. Edwin E. Graham, M.D., Philadelphia.

Auriculoventricular Heart Block in Children, with Report of a Case. J. A. E. Eyster and W. S. Middleton, Madison, Wis.

Abscess of the Lungs in Infants and Children. Harry Wessler, M.D., and Herman Schwarz, M.D., New York.

The Elimination of Acetone Bodies During Infectious Fevers. Borden S. Veeder, M.D., St. Louis, and Meredith R. Johnston, M.D., Captain, M. C., U. S. Army.

Clinical Department:
A Report of Two Cases of Congenital Stricture of the Esophagus. John Lovett Morse, A.M., M.D., Boston.

Progress in Pediatrics:
Recent Work in Anatomy, Physiology and Pathology of Infancy and Childhood. James B. Holmes, M.D., Baltimore.

MARCH 1920. NUMBER 3.

Dilatation of the Colon in Children. Alfred Edward Meyers, M.D., San Francisco.

Osteogenesis Imperfecta Congenita. H. M. McClanahan, M.D., and W. W. Willard, Ph.D., Omaha.

Calcium Metabolism of Infants and Young Children, and the Relation of Calcium to Fat Excretion in the Stools. Part 2.—Children Taking a Mixed Diet. L. Emmett Holt, M.D., Angelia M. Courtney and Helen L. Fales, New York.

Chondrodysplasia; Multiple Cartilaginous Exostoses. Report of Cases. Hugh L. Dwyer, M.D., Kansas City, Mo.

A Note on Renal Function in Scarlet Fever. Borden S. Veeder, St. Louis, and Meredith R. Johnston, Captain M. C., U. S. Army.

An Improved Needle for Sinus Therapy. Alton Goldbloom, M.D., New York.

Progress in Pediatrics:
Review of Literature on Respiratory Diseases. Louis C. Schroeder, M.D., New York.

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INFANT AND CHILD MORTALITY

INCLUDING MISCARRIAGES AND STILLBIRTHS *

HERMAN SCHWARZ, M.D.
NEW YORK

The material for this work is taken from the social histories of 10,000 families which we have gathered in the course of ten years. The social status represented is that usually seen in a large city such as New York, in families in which the earning capacity in prewar times was from ten to eighteen dollars a week, a bare living wage. The nationalities included are approximately:

	Per Cent.
Americans (born in the United States)	17
Russian Jews, native and foreign born	40
Austro-Hungarians	22
Negroes	5
Italians	5
Miscellaneous	11

We were fortunate in having, during the period covered, the same social worker, a woman versed in the various languages, so that the data may be considered fairly even and accurate. The social histories were obtained on special cards,[1] on which were noted income, nationality, pregnancies, miscarriages, etc. Of the 10,000 charts of social histories thus obtained, we have analyzed only the first 7,000, partly on account of the stupendous amount of work involved, and also because we believe that the present moment — when all the belligerent nations are anxious to do everything possible for the welfare of the coming generation — is an opportune time to publish some of our figures. We hope that any information which we have gathered may be of some value.[2]

Table 1 gives a general survey of the scope of this contribution to the study of infant mortality. In 6,968 families there were 27,711

* A statistical report from the Pediatric Department of the John E. Berwind Maternity Clinic.

1. See first annual report of the clinic published in 1908.

2. In order to make the tables as easily understood as possible, we would note that "total pregnancies" means all the pregnancies, including live born miscarriages and stillbirths. Living births refers to those pregnancies which resulted in live born infants.

pregnancies, including miscarriages and stillbirths, an average of 3.9 per family. Of these 27,711 pregnancies, 2,239 were miscarriages and 413 were stillbirths, the remainder, or 25,059, represent the number of living births; that is, there were 3.6 live born children to each family. The total number of deaths under 1 year of living births were 3,232. From the first to the eighth year there were 1,081 additional deaths, or a total of 4,313 deaths of living births. Taking all the deaths up to 8 years, including miscarriages and stillbirths, we find that of the 27,711 pregnancies, 6,965 babies, or 25 per cent., died; that is, wasted:

TABLE 1.—RESULTS OF GENERAL SURVEY OF INFANT MORTALITY

Number of families	6,968	
Number of pregnancies (miscarriages and stillbirths included)	27,711	
Number of miscarriages	2,239	
Percentage of miscarriages to total pregnancies		8.07
Number of stillbirths	413	
Percentage of stillbirths to total pregnancies		1.49
Total deaths under 1 year of living births	3,232	
Percentage of infantile deaths to total pregnancies		11.66
Percentage of infantile deaths to living births		12.89
Total deaths of infants and children up to 8 years	4,313	
Percentage of child deaths up to 8 years to total pregnancies		15.56
Percentage of child deaths up to 8 years to living births		17.21
All fatalities—deaths, stillbirths, miscarriages	6,965	
Percentage of all fatalities to total pregnancies		25.13
Miscarriage rate	80.7 per 1,000 (total) pregnancies	
Miscarriage rate	89.3 per 1,000 living births	
Stillbirth rate	14.9 per 1,000 (total) pregnancies	
Infant death rate	116.6 per 1,000 (total) pregnancies	
Infant death rate	128.9 per 1,000 living births	
Child death rate (to 8 years)	155.6 per 1,000 (total) pregnancies	
Child death rate (to 8 years)	172.1 per 1,000 living births	
Total fatalities rate	251.3 per 1,000 (total) pregnancies	

TABLE 2.—NUMBER OF PREGNANCIES PER FAMILY

Number of Families in Series	Number of Pregnancies per Family	Total Number of All Pregnancies	Number of Families in Series	Number of Pregnancies per Family	Total Number of All Pregnancies
1,057	1	1,057	57	11	627
1,276	2	2,552	43	12	516
1,177	3	3,531	15	13	195
1,029	4	4,116	16	14	224
779	5	3,895	5	15	75
607	6	3,642	4	16	64
405	7	2,835	3	17	51
239	8	1,912	3	19	57
168	9	1,512			
85	10	850	6,968		27,711

an entire economic loss. Unfortunately, our statistics cannot be used to total the amount of loss up to the working age in order to find out the real economic loss to the nation. Computing rates in terms of a thousand pregnancies, we find that the miscarriage rate in these families was 80.7 per thousand of all pregnancies and 89.3 per thousand of living births. The infant death rate (up to 1 year of age) in this group of families was 128 per thousand living births. A contrast to this figure is the death rate of 70 per thousand in this same group of families of only those of their children who, during the ten years,

were under our care. This figure, which shows a saving of 58 infants per thousand living births, proves conclusively what supervision, education and care can accomplish. The child death rate (up to eight years) reached 172 per thousand. In other words, 172 out of every thousand children born alive, died before they reached the age of 8 years.

TABLE 3.—AVERAGE NUMBER OF MONTHS BETWEEN PREGNANCIES

Number of Families in Series	Number of Pregnancies per Family	Total Number of Pregnancies	Average Number of Months between Pregnancies
1,276	2	2,552	15
1,177	3	3,531	19
1,029	4	4,116	22.5
779	5	3,895	22.5
607	6	3,642	23
405	7	2,835	22
239	8	1,912	21
168	9	1,512	21
85	10	850	20.5
57	11	627	18
43	12	516	20
15	13	195	19
16	14	224	16.5
5	15	75	15
4	16	64	13
3	17	51	13.5
3	19	57	12.5
5,911		26,654	20

In Table 3 we have tried to show how frequently pregnancy occurs in these families. As the marriage date was not available in our data, and the length of marriage thus impossible to obtain, we computed the months between the various pregnancies from the date of the termination of the first pregnancy. It will be seen that the average interval between pregnancies, in families with from four to six pregnancies, is twenty-two and a half months, and in families with from two to ten pregnancies the average interval in never less than twenty months. Even among these families there is not a child born every year. From this standpoint it might not be inappropriate to say a word concerning the declining birth rate. Since so much vicious propaganda has been going on in all countries about birth control I cannot do better than to refer to Louis I. Dublin's address as retiring vice president of the American Association for Advancement of Science, read at Pittsburgh, Dec. 29, 1917, Section I, Social and Economic Science. He showed that at the beginning of the nineteenth century, France had a population of 29,000,000; Germany a population of 23,000,000, and England and Wales a population of 18,000,000. At the beginning of the twentieth century the facts were almost reversed. Germany's population was 65,000,000; England and Wales, 45,000,000, and France 39,000,000. In 1860, France was still in the lead with a population of 37,000,000; from 1860 to 1914 it increased only 2,000,000. This was in part due to

decreased birth rate, but also to a high infantile death rate. In 1830, the birth rate in France was 30 per thousand; in 1914 it was 18 per thousand, and the infantile death rate was 19.6 per thousand — the death rate exceeding the birth rate. To show, as Mr. Dublin does so well, how this shifts the population, it might be interesting to know that in Germany, one quarter of the population was less than 11 years of age; in England and Wales, one quarter of the population was less than 12 years of age; in France, one quarter of the population was less than 14 years of age. In Germany, three quarters of the population was less than 41 years of age; in England and Wales, three quarters of the population was less than 42 years of age, and in France, three quarters of the population was less than 49 years of age. In the United States, although our statistics are not very accurate, the birth rate is probably 25 per thousand, and the death rate 15 per thousand, a yearly increase of about 1 per cent. to our population.

TABLE 4.—INCIDENCE OF MISCARRIAGE

Total Number of Families in Series	Number of Pregnancies per Family	Number of Families in which Miscarriage Occurred	Total Number of Miscarriages	Total Number of All Pregnancies	Miscarriages per Thousand of All Pregnancies
1,057	1	0	0	1,057	0.0
1,276	2	93	94	2,552	36.8
1,177	3	155	162	3,531	45.8
1,029	4	209	260	4,116	63.4
779	5	225	288	3,895	72.6
607	6	222	306	3,642	83.4
405	7	158	257	2,835	91.0
239	8	125	231	1,912	120.8
168	9	110	211	1,512	139.5
85	10	55	120	850	141.2
57	11	34	71	627	113.2
43	12	33	78	516	151.1
15	13	14	46	195	235.8
16	14	12	40	224	178.5
5	15	5	23	75	306.6
4	16	4	20	64	312.5
3	17	3	17	51	333.3
3	19	3	20	57	350.8
6,968	..	1,460	2,239	27,711	80.7

Percentage of miscarriages to total pregnancies, 8.07.
Percentage of miscarriages to living births, 8.92.

It may be asked, "How large must a family be in order to keep our population from diminishing?" In order to have an individual born, there certainly must be two others alive. If we take 100,000 individuals at birth, only 73,000 (Dublin) are alive at the age of marriage,[3] so that at least two and one half individuals are necessary to produce another individual. At least from 12 to 15 per cent. of these 73,000 do not marry, and 7 per cent. of those who do marry have no children, so that from three and one half to four individuals must be born to insure a new individual.

3. According to our figures many less than this are alive at the marriageable age.

In 6,968 families with 27,711 pregnancies, including living births, miscarriage and stillbirth pregnancies, there were 2,239 miscarriages, a rate of 80.7 per thousand, or 8 per cent. of all the pregnancies. This miscarriage rate is in direct proportion to the number of pregnancies in the family. With two pregnancies per family, the rate was 36.8 per thousand; with three pregnancies, it was 45.8 per thousand, and so on, until in eighty-five families with ten pregnancies, the rate was 141.2 per thousand, more than 10 per cent. of all the pregnancies ending in miscarriages. Of the 850 pregnancies in families with more than ten pregnancies per family, the number is really too small to make any definite statement, yet it is interesting to see, from what material we have, how great the fetal death rate is. For instance, in sixteen families with fourteen pregnancies each, i. e., 224 pregnancies, there were forty miscarriages, or 17 per cent. In four families with sixteen pregnancies each, the fetal death rate was 30 per cent.

TABLE 5.—INCIDENCE OF STILLBIRTHS

Total Number of Families in Series	Number of Pregnancies per Family	Number of Families in which Stillbirths Occurred	Total Number of Stillbirths	Total Number of All Pregnancies	Stillbirths per Thousand of All Pregnancies
1,057	1	0	0	1,057	0.0
1,276	2	22	22	2,552	8.6
1,177	3	42	45	3,531	12.7
1,029	4	48	51	4,116	12.3
779	5	59	63	3,895	18.9
607	6	46	58	3,642	15.9
405	7	33	43	2,835	15.2
239	8	32	43	1,912	22.4
168	9	17	25	1,512	16.5
85	10	23	26	850	30.5
57	11	10	18	627	27.5
43	12	6	12	516	23.2
15	13	3	6	195	30.7
16	14	0	0	224	0.0
5	15	1	1	75	13.3
4	16	0	0	64	0.0
3	17	0	0	51	0.0
3	19	0	0	57	0.0
6,968	..	342	413	27,711	14.9

Percentage of stillbirths to total pregnancies, 1.49.
Percentage of stillbirths to living births, 1.64.

The stillbirth rate per thousand total pregnancies was 14.9, and per thousand living births it was 16.4. This is much lower than that for New York City, which, according to Dr. Guilfoy,[4] was from 43 to 49 per thousand births during the past five years. The same condition as in the miscarriage rate exists here also, the rate going up steadily in direct proportion to the number of pregnancies in the family. With two pregnancies per family, the rate was 8.6 per thousand of all pregnancies; with ten pregnancies per family, the rate was 30.5 per thousand. The total still birth rate is about 14.9 per thousand preg-

4. Personal communication to the author.

nancies, or 1.49 per cent. This is more than the stillbirth rate in the various cities, but I believe it to be more accurate, for many stillbirths are undoubtedly not reported.

Table 6 gives a very good idea of the relation of the infant mortality rate to the entire pregnancy rate and to the "living births" rate, if I may use that term. Here, again, there is a direct relationship between the number of pregnancies of all kinds and also the number of living births to the infant mortality rate. With two pregnancies in the family, the rate is 75.1 and 78.4 per thousand, respectively, whereas with ten pregnancies, the rate is 174.1 and 210.2 per thousand. Families with a greater number of pregnancies are interesting only from the standpoint of the greater death rate present. The average infant mortality rate in all of our families was 116.6 per thousand for all pregnancies, and 128.9 per thousand for living births, whereas, under the same living and social conditions, but under the supervision of our clinic, the death rate for living births in these families has never been more than 70 per thousand.

TABLE 6.—DEATHS OF LIVE BORN INFANTS FROM BIRTH TO ONE YEAR OF AGE. INFANT MORTALITY

Total Number of Families in Series	Number of Pregnancies per Family	Number of Families in which Death Occurred (Birth–1 Year)	Total Number of Deaths of Infants	Total Number of All Pregnancies	Deaths per Thousand of All Pregnancies	Total Number of Living Births	Deaths per Thousand of Living Births
1,057	1	57	57	1,057	53.9	1,057	53.9
1,276	2	177	191	2,552	75.1	2,436	78.4
1,177	3	275	314	3,531	88.9	3,324	91.7
1,029	4	308	372	4,116	90.3	3,805	97.7
779	5	310	413	3,895	106.0	3,549	116.3
607	6	314	471	3,642	129.3	3,278	143.9
405	7	246	375	2,833	132.2	2,535	143.9
239	8	149	267	1,912	139.6	1,638	162.6
168	9	119	245	1,512	162.0	1,276	192.7
85	10	66	148	850	174.1	704	210.2
57	11	48	137	627	218.5	538	254.6
43	12	35	102	516	191.8	426	239.4
15	13	13	26	195	133.3	143	181.8
16	14	16	61	224	272.4	184	331.5
5	15	4	13	75	173.3	51	254.9
4	16	3	16	64	250.0	44	363.6
3	17	3	18	51	352.9	34	529.4
3	19	2	6	57	105.2	37	162.1
6,968	..	2,145	3,232	27,711	116.6	25,059	128.9

Percentage of infant deaths to total pregnancies, 11.66.
Percentage of infant deaths to living births, 12.89.

Table 7 shows still better what happens to children in these families, especially in relation to the number of children in the family. With two and three pregnancies the table does not really describe conditions accurately, for many of the children born had not reached the age of 8 years. However, in going up the scale, it will be seen that with five pregnancies per family the death rate is 162.8 per thousand, or 16 per cent. of all the children born alive never reach the age of 8 years

With ten pregnancies, 29 per cent. of those born alive succumb before reaching the eighth year. There were 25,059 living births with 4,313 deaths, a mortality of 17 per cent. Of these deaths, 3,232 occurred during the first year and 1,081 children died between the first and the eighth year.

TABLE 7.—DEATH OF CHILDREN FROM BIRTH TO EIGHT YEARS OF AGE

Total Number of Families in Series	Number of Pregnancies per Family	Number of Families in which Death Occurred (Birth–1 Year)	Total Number of Deaths of Children	Total Number of All Pregnancies	Deaths per Thousand of All Pregnancies	Total Number of Living Births	Deaths per Thousand of Living Births
1,057	1	57	57	1,057	53.9	1,057	53.9
1,276	2	200	213	2,552	83.4	2,436	87.4
1,177	3	331	378	3,531	104.3	3,324	113.7
1,029	4	407	502	4,116	121.9	3,806	131.9
779	5	413	578	3,895	148.4	3,549	162.8
607	6	393	644	3,642	177.4	3,278	196.4
405	7	294	523	2,835	184.4	2,535	206.3
239	8	182	377	1,912	171.5	1,638	200.2
168	9	137	328	1,512	216.9	1,276	257.0
85	10	73	205	850	241.1	704	291.1
57	11	53	190	627	303.0	538	353.1
43	12	43	139	516	269.3	426	326.2
15	13	14	36	195	184.6	143	251.7
16	14	16	74	224	375.0	184	402.1
5	15	5	16	75	213.3	51	313.7
4	16	4	19	64	296.8	44	431.8
3	17	3	23	51	450.9	34	655.8
3	19	2	11	57	192.9	37	297.1
6,968	..	2,627	4,313	27,711	155.6	25.059	172.1

Percentage of total deaths of children up to 8 years to total pregnancies (miscarriages and stillbirths), 15.56.
Percentages of total deaths of children up to 8 years to living births, 17.21.

TABLE 8.—ALL FATALITIES—DEATHS, MISCARRIAGES, STILLBIRTHS—IN FAMILIES UP TO EIGHT YEARS

Total Number of Families in Series	Number of Pregnancies per Family	Number of Families in which Fatalities Occurred	Total Number of Fatalities	Total Number of Pregnancies	Fatalities (Living Births, Miscarriages and Stillbirths) per Thousand of All Pregnancies
1,057	1	57	57	1,057	53.9
1,276	2	298	329	2,552	128.9
1,177	3	487	585	3,531	165.6
1,029	4	570	813	4,116	197.5
779	5	537	924	3,895	237.2
607	6	475	1,008	3,642	277.0
405	7	342	823	2,835	290.3
239	8	216	651	1,912	340.4
168	9	167	564	1,512	373.0
85	10	78	351	850	412.9
57	11	57	279	627	445.9
43	12	43	229	516	443.7
15	13	15	88	195	451.2
16	14	16	114	224	508.9
5	15	5	40	75	533.3
4	16	4	39	64	609.3
3	17	3	40	51	784.3
3	19	3	31	57	561.4
6,968	..	3,383	6,965	27,711	251.3

Percentages of all fatalities to all pregnancies, 25.13.

TABLE 9.—Infant and Child Mortality (Up to Eight Years) in Relation to the Nationality of the Parents.
Material: 5,968 Families; 26,711 Pregnancies

	Number of Families	Total Number of Pregnancies	Number of Living Births	Number of Live Children per Family	Number of Deaths of Living	Rate of Death per Thousand Total Pregnancies	Rate of Death per Thousand Living births	Number of Miscarriages	Miscarriage Rate per Thousand Total Pregnancies	Miscarriage Rate per Thousand Living Births	Number of Stillbirths	Stillbirth Rate per Thousand Total Pregnancies	Stillbirth Rate per Thousand Living Births	Total Number Fatalities	All Fatalities per Thousand Total Pregnancies
Both parents born in U. S. A., largely second generation of foreign extraction	807	3,567	3,131	3.8	787	220.6	251.2	374	104.9	119.4	62	17.4	19.7	1,223	342.8
Father born in U. S. A.	210	885	785	3.7	172	190.4	219	76	85.7	96.8	24	27.1	30.0	272	307.3
Mother born in U. S. A.	174	773	698	4.0	149	192.7	213.4	62	80.2	88.8	13	16.7	18.6	224	289.7
Both parents of foreign birth	4,525	20,353	18,477	4.1	3,007	142.4	162.7	1,595	77.9	86.2	281	13.3	15.2	4,883	240
Parents colored; born in U. S. A. and British West Indies	250	1,120	957	3.7	196	175	204.8	130	116	135.8	33	29.5	34.3	359	320.5

MISCARRIAGE RATE TO ALL PREGNANCIES AND LIVING BIRTHS UP TO THE EIGHTH YEAR

	Deaths per Thousand Total Pregnancies	Deaths per Thousand Living Births
Both parents born in the U. S. A.	104.9	119.4
Father born in the U. S. A.	76	85.7
Mother born in the U. S. A.	62.8	80.7
Both parents of foreign birth	79.9	86.2
Colored group	116	135.8

DEATH RATE OF ALL FATALITIES TO THE TOTAL PREGNANCIES UP TO THE EIGHTH YEAR

	Deaths per Thousand of All Pregnancies	
	Number	Percentage
Both parents born in the U. S. A.	342.8	34
Father born in the U. S. A.	307.8	30.7
Mother born in the U. S. A.	289.7	28.9
Both parents of foreign birth	240	24
Colored group	320.5	32

STILLBIRTH RATE TO ALL PREGNANCIES AND LIVING BIRTHS UP TO THE EIGHTH YEAR

	Deaths per Thousand Total Pregnancies	Deaths per Thousand Living Births
Both parents born in the U. S. A.	17.4	19.7
Father born in the U. S. A.	27.1	30
Mother born in the U. S A.	16.7	18.6
Both parents of foreign birth	13.3	15.2
Colored group	29.5	34.3

DEATH RATE OF LIVE BORN CHILDREN TO TOTAL PREGNANCIES AND LIVING BIRTHS UP TO THE EIGHTH YEAR

	Deaths per Thousand Total Pregnancies	Deaths per Thousand Living Births
Both parents born in the U. S. A.	220.6	251.2
Father born in the U. S. A.	190.4	219
Mother born in the U. S. A.	192.7	213.4
Both parents of foreign birth	142.4	162.7
Colored group	175	204

TABLE 10.—Fatalities in Relation to Literacy of Parents Up to Eight Years. Materials 5,968 Families; 26,711 Pregnancies

	Number of Families	Total Number of Pregnancies	Number of Living Births	Number of Deaths of Living Births	Deaths of Living Births per Thousand Total Pregnancies	Deaths of Living Births per Thousand Living Births	Number of Miscarriages	Miscarriage Rate per Thousand Total Pregnancies	Miscarriage Rate per Thousand Living Births	Number of Stillbirths	Stillbirth Rate per Thousand Total Pregnancies	Stillbirth Rate per Thousand Living Births	Number of Total Fatalities	Total Death per Thousand Total Pregnancies	Total Death per Thousand Living Births
One or both parents literate	4,897	22,298	20,050	3,513	112.5	125.3	1,906	85.5	95	342	15.3	17	5,761	258	175.2
Both parents illiterate	966	3,870	3,513	713	184.5	203.1	291	75.1	82.4	66	17	18.7	1,070	276.4	202.9

FATALITIES OF ALL PREGNANCIES UP TO THE EIGHTH YEAR

	Deaths per Thousand Living Births	Deaths per Thousand Total Pregnancies
One or both parents literate	17.5%	25.8%
One or both parents illiterate	20.2%	27.6%

MISCARRIAGE RATE

	Miscarriages per Thousand Living Births	Miscarriages per Thousand Total Pregnancies
One or both parents literate	85.5	95+
One or both parents illiterate	75	82.4

FATALITIES OF LIVING BIRTHS UP TO THE EIGHTH YEAR

	Deaths per Thousand Living Births	Deaths per Thousand Total Pregnancies
One or both parents literate	11.2%	12.5%
One or both parents illiterate	18.4%	20.3%

STILLBIRTH RATE

	Stillbirths per Thousand Living Births	Stillbirths per Thousand Total Pregnancies
One or both parents literate	15.3	17
One or both parents illiterate	17	18.7

Table 8 shows all fatalities in these families up to 8 years. Here, again, the relationship to the number of pregnancies is apparent, and, in addition, the important economic fact is shown that 25 per cent. of all the conceptions have been for naught before first eight years of life are passed.

The nationality of the parents in relation to infant and child mortality is perhaps a minor factor, for poverty, housing conditions and literacy all form a part of the great cause of child mortality. Yet, it is the common experience of those who have worked among the poor to find that the native born who have been content to remain in the slums do so because they are often a very shiftless group, and who, above all, either do not seem to have the ability or the desire to nurse their children. The death rate, in early infancy, of children of native born parents, greatly exceeds that of the foreign element. It is well known that the nursing infant is fairly immune to its surroundings as long as it is kept on the breast. Thus, the figures for total fatalities of 342, 307 and 289 per thousand pregnancies of native born parents, as compared with 240 per thousand born of foreign parents, are not surprising. The fatalities in the live born children are just as marked. This also seems to hold good in other strata of society, for in the 1916 census of the births and deaths in the registration area the same thing is observed. The colored group, curiously enough, shows less total fatalities than in the group in which both parents (white) were born in the United States. The number of our colored families is too small, however, to make satisfactory comparisons.

The miscarriage rate is greatest when both parents are native born and least when both parents are foreign born. The colored group, as was to be expected, shows the largest number of miscarriages, owing, no doubt, to the great amount of syphilis present.

The stillbirth rate is made up of so few cases that it is best not to draw any deductions.

The relation of infant and child mortality to the literacy of the parents is always interesting, but very difficult to make convincing. That parents who are literate are apt to have a more systematic and well regulated household there can be no doubt; that literacy tends to a clearer comprehension of the instructions given and thus an increased knowledge of infant and child welfare, there is no question. Yet shiftlessness and drunkenness can very well go along with literacy. However, literacy in the parents does place them on a little higher plane of civilization than illiteracy. The table shows very nicely the difference in the two classes, and very much in favor of literacy.

The summaries of Table 10 show that of the deaths of living births, the rate for literacy is 112.5 to 125.3 as compared with 184 and 203.1, respectively, for illiteracy. In the miscarriage rate, however, the con-

ditions are reversed, for the literate have more miscarriages than the illiterate. In the stillbirth rate, the figures are about the same, which might be expected. In a previous tabulation[5] we showed a similar condition, but with fewer families. There were 358 literate families with an infantile mortality rate of 111 per thousand born, and 113 illiterate families with an infant mortality rate of 172 per thousand born. It does seem that education is a very important factor.

We wish to thank Dr. I. L. Hill and Mr. John E. Berwind for the hearty cooperation and advice which they have given us.

5. First Annual Report of the John E. Berwind Maternity Clinic.

RESULTS OF SOME EXPERIMENTAL WORK WITH SODIUM CACODYLATE ON ATHREPTIC INFANTS

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OMAHA

The physician is rather frequently confronted with a type of infant, who, because of some nutritional disturbance, either fails to gain in weight, or the gain is so slight as to be almost negligible.

This condition of insufficient nutrition is sometimes called athrepsia. The condition is prevalent in hospitals or institutions where many babies are housed. However, it is not uncommon in private practice and among the well-to-do. Its occurrence is much more frequent in the artificially fed infant, although it may be encountered occasionally in the breast fed.

One member of this class of infants will take its food well, does not vomit and there is no indication of digestive disturbance. The food may be of sufficient caloric value. Another member of this class of cases, will tolerate only a certain amount of food. In feeding these infants, one has to be extremely careful not to overtax this food tolerance. They are particularly unable to digest any normal amount of fat. If one exceeds their food tolerance, there occurs a definite gastro-intestinal upset, with vomiting, usually a diarrhea and more rapid loss of weight.

There is in both types of cases, an inability to assimilate food properly, so as to make a normal gain in weight, in spite of careful regime as to food, time and regularity of feeding.

Careful manipulation of the food, even resorting to breast feeding, makes very little, if any, change in the infants. They do not do well. They are below par physically, anemic, and compare very unfavorably with normal, healthy infants of the same age.

In groping about for a possible solution of this problem, it seemed to one of us that the administration of arsenic to these infants might so improve them physically as to enable them to take on more weight. Spencer L. Dawes and H. C. Jackson, in a series of experiments on dogs and rabbits, concluded that after the injection of sodium cacodylate, arsenic, in inorganic form, makes its appearance in the tissues of the body. The animal body possesses the power to reduce or decompose the organic arsenic compound, sodium cacodylate, into the inorganic active arsenous or arsenic acid. Cacodyl or some of its salts are also found in the tissues after injections of sodium cacodylate, but they are of little or no therapeutic value. It is admitted that a

considerable quantity of the inorganic arsenic is eliminated by the urine, and probably by the sweat and breath; but enough is retained to have some effect therapeutically.

In their experiments, Dawes and Jackson note that almost invariably after injections the patient gains in weight. In anemic cases the percentage of hemoglobin is always increased. Arsenic exerts a tonic effect, the nutrition improving, and even in health the individual taking it in carefully graduated doses, takes on more flesh, and is not fatigued readily.

In endeavoring to come to some conclusion in this class of cases six infants were selected for study. They were all institution babies. The oldest child had been in the institution one year. Of the others, two children had been in for five months, two for six months, and child one for four months. They were all very much under weight and more or less anemic. They were all athreptics.

Before beginning the injections, a von Pirquet was done on each one with negative results. A blood Wassermann from each infant proved negative. A routine urine examination yielded similar negative results.

Regarding the diet: One child was taking cooked cereals, broths, toast, cooked vegetables, custards, jellies and about one pint of milk in twenty-four hours. Two children were taking a modified milk formula of about the same caloric value. One child was taking breast milk exclusively. One was on partial breast milk and modified milk; one was taking buttermilk with approximately a 10 per cent. corn syrup. Those children taking a bottle or breast were fed every three hours, seven feedings in twenty-four hours. The little girl on mixed diet was fed four times daily. The feedings were not changed during the course of the injections.

At the time of beginning injections, one child was 15 months old; two children were 9 months old; three were 6 months old.

Eight injections were given in all, at intervals of four days. All injections were given intramuscularly and hypodermically. The youngest infant received ¼ grain, as an initial dose; the oldest ¾ grain. The dosage was gradually increased till the youngest was receiving ¾ grain and the oldest 1 grain. The maximum dose at any one time was 1 grain.

A blood examination was done on all the infants before the injections were begun. A second examination was done after two injections, and a third after the last injection. The blood examination included a hemoglobin estimation, red and white cell count, together with a differential leukocyte count. The red and white cell count remained nearly uniform throughout, nor was there any marked change in the differential count. There was, however, a striking

increase in the hemoglobin in all the children. Three children showed an increase of five points; one child of ten points; one twenty points, and one twenty-five points. This increase in hemoglobin has been noted by other observers.

One child failed to gain in weight during the injections, but in the month succeeding gained 1 pound, 10 ounces. Three children gained approximately 2 pounds, and two gained 1⅓ pounds. These gains have been continued since stopping the treatment. They all improved in nutrition and seemed to be more hungry for their food when feeding time arrived.

From the foregoing, it may be possible to draw the following conclusions:

1. That carefully graduated doses of sodium cacodylate, when injected hypodermically into infants, has no toxic influence.

2. The percentage of hemoglobin in the blood is uniformly increased.

3. While the series of cases is not large, all the children showed a substantial gain in weight, and one may conclude that sodium cacodylate possesses tonic properties enabling the individual athreptic infant to take on weight, and possibly enable him to assimilate his food more properly and thus improve his nutrition.

1. Handbook of Medical Science, New York, Wm. Wood & Co., **1**:636.
2. Hare's Practical Therapeutics, Ed. 16, Philadelphia, Lea & Febiger, p. 120.
3. Sollmann: Manual of Pharmacology, Philadelphia, W. B. Saunders Co., p. 617.
4. Forchheimer: Therapeusis of Internal Diseases, New York, D. Appleton & Co. **4**:248.
5. Dawes, S., and Jackson, H. C.: J. A. M. A. **48**:2090, 1907.

PROGNOSIS IN OPERATED CASES OF HYPERTROPHIC STENOSIS OF PYLORUS *

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One hundred and sixty-three Rammstedt operations were performed for hypertrophic stenosis of the pylorus at the Babies Hospital between Jan. 1, 1915, and July 1, 1919. In this number are included cases already reported by Holt,[1] Downes[2] and Kerley,[3] together with a number of cases not yet published. Holt made a survey of the whole subject; compared the medical with the surgical treatment, and demonstrated the superiority of the Rammstedt operation over other methods of treatment previously employed in this hospital. Downes, who performed the majority of the operations, described in detail the operative technic. Kerley reports twenty-six cases. These are included in this series. We have grouped all these cases together for the purpose of studying them from the standpoint of prognosis.

One hundred and thirty-one children recovered and thirty-two died, a mortality of 19.63 per cent. It is interesting to note, in connection with many previous observations, that 133 were boys and only 30 were girls. Of the boys, twenty-nine died, a mortality of 21.8 per cent., and of the girls, three died, a mortality of 10 per cent. These latter figures, however, are hardly significant.

The general rule followed at the hospital for the last few years, has been to operate as soon as a definite diagnosis has been made. Many of these children were in extremely bad condition on admission as a result of the prolonged vomiting, but in only one instance in the last six years has an operation been refused because of the child's condition. This baby had been vomiting for eight weeks, weighed less than 4 pounds, and lived only a few hours after entering the hospital. Deaths from inanition, three or four weeks after operation, are counted as operative deaths.

It becomes evident from a study of our series that the operation per se is, perhaps, the least important factor in the mortality. The condition of the baby at the time of operation is certainly the most important factor.

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* From the Babies' Hospital, New York, N. Y.

1. Holt, L. E.: J. A. M. A. **68**:1517 (May 26) 1917.
2. Downes, W. A.: Surg. Gyn. & Obst. **22**:251 (March) 1916.
3. Kerley, G. G.: J. A. M. A. **72**:16 (Jan. 4) 1919.

CAUSES OF DEATH

Fourteen babies died from collapse from one to three days after operation, and five died from general peritonitis. In one of the latter cases the duodenum was known to have been opened accidentally at the time of operation. One child died of bronchopneumonia complicating whooping cough, and one child died from postoperative hemorrhage on the third day after operation. In this last case there was a definite history of bleeding in the family. The remaining eleven deaths were from marasmus and occurred in from three to twenty-five days after operation. In all cases, then, except the seven in which death was due to complications, the fatal result can definitely be attributed to a state of inanition existing at the time of operation. It is true, however, that the extra load incident to the anesthetic and to the shock of the operation contributed to the fatal termination. It is no less true, that operation in these cases offered the only chance of recovery.

TABLE 1.—DURATION OF SYMPTOM AND MORTALITY

Duration of Symptoms	Cases	Deaths	Mortality, %
1 week or less	20	1	5.0
1 – 2 weeks	38	7	18.42
2 – 3 weeks	33	2	6.66
3 – 4 weeks	24	5	20.83
4 – 5 weeks	19	6	31.58
More than 5 weeks	29	11	38.28
SUMMARY			
Under 4 weeks	115	15	13.04
More than 4 weeks	48	17	35.42

TABLE 2.—PREVIOUS FEEDING

	Cases	Deaths	Mortality, %
Breast fed	106	12	11.3
Artificially fed	57	20	35.0

In considering the factors which influence the prognosis, we have studied them under the following headings: (1) The duration of symptoms before operation; (2) the type of feeding before operation; (3) the weight at the time of operation; (4) the percentage of weight loss at the time of operation.

In Table 1 is shown the mortality in relation to duration of symptoms before operation. Since vomiting is usually the first symptom noted by the mother, we have taken this as the basis of our time divisions. As shown by this table, the mortality increased directly with the duration of symptoms, except in those children who vomited from one to two weeks before operation. Of these, thirty-one recovered and seven died, a mortality of 18.42 per cent. This is much higher than the mortality in the group in which vomiting occurred from two to three weeks before operation. In examining the records in

the seven fatal cases in this group, it was found that there were two deaths from general peritonitis and five from inanition. All of the children suffering from inanition had lost 25 per cent. or more of their weight even though their symptoms had lasted less than two weeks. Comparing these with the fatalities in the other groups it was evident that this group contained a larger proportion of severe cases.

The mortality as influenced by the type of feeding before operation is shown in Table 2. In determining the type of feeding, we have considered those artificially fed who had received no breast milk during the two weeks preceding their admission to the hospital. Infants only partially artificially fed were classed as breast fed, as also were those weaned within the two weeks preceding their admission. In these latter cases, the mothers' milk had not entirely disappeared and was often easy to reestablish.

TABLE 3.—PREVIOUS FEEDING, AND DURATION OF SYMPTOMS

	Cases	Deaths	Mortality, %
Breast Fed Babies:			
Vomiting, less than 4 weeks	88	9	10.2
Vomiting, more than 4 weeks	18	3	16.6
Artificialy Fed Babies:			
Vomiting, less than 4 weeks	27	6	22.2
Vomiting, more than 4 weeks	30	14	46.6

TABLE 4.—BODY WEIGHT AT TIME OF OPERATION

Weight	Cases	Deaths	Mortality, %
5 pounds or less	5	2	40
5 - 6 pounds	32	10	31
6 - 7 pounds	57	14	25
7 - 8 pounds	45	5	11
8 - 9 pounds	18	1	6
More than 9 pounds	6	0	0
SUMMARY			
Less than 7 pounds	94	26	28
7 pounds and over	69	6	8.7

In order to determine whether the mortality based on the duration of symptoms was influenced in any way by the type of feeding, we have divided the breast and the artificially fed cases, each, into two groups which correspond to the summary of Table 1. Namely, (1) those children who had vomited less than four weeks and (2) those who had vomited four weeks and more.

A study of Table 3 shows that the mortality in the corresponding divisions is definitely lower for the breast fed infants. It is interesting to observe that the mortality of the breast fed infants who had vomited more than four weeks is lower than that of the artificially fed infants who had vomited less than four weeks.

The weight at the time of operation has always been considered a valuable guide in determining the prognosis. Table 4 shows the mortality based on this factor. The summary of Table 4 shows the enor-

mous difference in mortality between the infants weighing less than 7 pounds at the time of operation and those weighing 7 pounds and over.

The influence both of the duration of symptoms before operation and of the weight of the infant at the time of operation to the mortality is shown in Table 5. It is worthy of note, that an infant weighing 7 pounds and over who has vomited four weeks or more, has little better chance of recovery than the infant weighing less than 7 pounds who has vomited less than four weeks before coming to operation. The duration of the symptoms before operation, then, is a very important factor in influencing the mortality as well as is the weight of the infant at the time of operation.

TABLE 5.—WEIGHT AND DURATION OF SYMPTOMS

	Cases	Deaths	Mortality, %
Vomiting less than 4 weeks:			
Weight 7 pounds and more	50	3	6.0
Weight less than 7 pounds	65	12	18.5
Vomiting 4 weeks and over:			
Weight 7 pounds and more	19	3	15.8
Weight less than 7 pounds	29	14	48.2

TABLE 6.—WEIGHT LOSS AND MORTALITY

	Cases	Deaths	Mortality, %
Total Cases:			
0 to 10 per cent. loss	26	0	0
10 to 20 per cent. loss	50	5	10
20 to 30 per cent. loss	39	13	33
More than 30 per cent. loss	12	6	50
Breast Fed:			
0 to 10 per cent loss	18	0	0
10 to 20 per cent. loss	33	1	3
20 to 30 per cent. loss	28	8	28.5
More than 30 per cent. loss	2	0	0
Artificially Fed:			
0 to 10 per cent. loss	8	0	0
10 to 20 per cent. loss	17	4	23.5
20 to 30 per cent. loss	11	5	45.5
More than 30 per cent. loss	10	6	60

Table 6 shows the mortality as influenced by the percentage of weight lost in the total number of cases, the breast fed cases and the artificially fed cases. There were 127 cases in which the actual loss in weight was known. Among these there were twenty-four deaths. In the entire group the mortality increased directly with the percentage of weight loss. This is to be expected from our findings in Table 4, which shows the mortality as influenced by the weight at the time of operation. Dividing the cases into two groups, namely, those children that had lost less than 20 per cent. of their best weight and those who had lost 20 per cent. and more of their best weight, there are in the former group seventy-six cases and five deaths, a mortality of 6.58 per cent., while in the latter group there are fifty-one cases with nineteen deaths, a mortality of 37.25 per cent.

There were eighty-one breast fed infants in the breast fed group of whom seventy-two recovered and nine died; a mortality of 11.11 per cent. There were only two breast fed infants who had lost 30 per cent. or more of their best weight; both recovered. Of course, no conclusion can be drawn from two cases. Forty-one breast fed infants came to operation who had lost less than 20 per cent. of their best weight; only one of these babies died. In this group are included the babies suffering from the milder form of this disease. Thirty breast fed infants had lost 20 per cent. and more of their best weight; eight of these died, a mortality of 26.66 per cent.

It is interesting to compare the figures of the breast fed infants in Table 6 with those of Table 3. In Table 3 there are eighty-eight breast fed infants in the group that vomited less than four weeks with seventy-nine recoveries; nine died, a mortality of 10.22 per cent. Table 6 shows that of the infants who had lost less than 20 per cent. of their best weight, forty recovered and only one died. Only one breast fed infant died, then, who had lost less than 20 per cent. of its body weight. It so happens that this infant was the one previously noted who died of postoperative hemorrhage, and in whose family there was a history of bleeding.

The total mortality for the artificially fed cases shown in Table 6 is 32.6 per cent. Twenty-five of these children had lost at the time of operation less than 20 per cent. of their best weight; there were four deaths, a mortality of 16 per cent. Twenty-one had lost 20 per cent. and more of their best weight, with eleven deaths, a mortality of 52.38 per cent. The advantage of breast feeding is very evident when the corresponding divisions in this group and in the artificially fed group (Table 6) are compared.

On an average, the ninety-four breast fed infants who recovered remained in the hospital after operation for 14.7 days; while with the thirty-seven artificially fed babies the average was 23.78 days. A breast fed baby recovering without any complications is usually discharged as soon as the wound is healed and the sutures are removed. As breast milk is used exclusively in the postoperative feeding, some additional time is required to put the artificially fed babies on a suitable formula. Hence, the necessity for a longer stay in the hospital.

The prognosis is greatly influenced by the postoperative care in these cases. The postoperative management as practiced in the Babies' Hospital has been published by E. A. Morgan.[4] The routine has varied only slightly since the publication of this paper. Breast milk is indispensable. At first, very small quantities of diluted breast milk are given, and increases in amount and strength are made very

4. Morgan, E. A.: Am. J. Dis. Child. **11**:245 (April) 1916.

gradually. As it is necessary to measure very accurately and increase very carefully the amount of food given, the baby is not allowed to nurse until a week after the operation. Gastric and intestinal disturbances are easily produced in these delicate infants. Their intestines through prolonged starvation are often unable to handle large quantities of food, even though that food be breast milk. The slightest tendency to vomiting and loose stools must be regarded with grave concern. We have found it advisable for the first few days after operation to pass the stomach tube immediately before each feeding. This aids in the expulsion of gas and mucus and adds to the comfort of the baby. In the event of vomiting, the quantity of food should be reduced temporarily, and an occasional gastric lavage should be given with 1 per cent. sodium bicarbonate solution. If the stool becomes loose, protein milk is used for a day or two instead of breast milk.

CONCLUSIONS

1. The duration of symptoms prior to operation is probably the most important single factor affecting the prognosis. When symptoms have lasted less than four weeks, the mortality is only one third as great as when they have lasted four weeks or longer.

2. The mortality in artificially fed babies is more than three times that for the breast fed babies.

3. In infants weighing 7 pounds or less, the mortality was three and one-half times as great as in those who weighed more than 7 pounds.

4. The mortality increases in direct proportion to the amount of weight lost previous to operation.

5. The mortality for breast fed infants who had vomited less than four weeks and who had lost less than 20 per cent. of their best weight is almost nil. The fatalities which occur are due to accidents usually avoidable when the operation is done by a skillful surgeon.

We wish to express thanks to Dr. L. Emmett Holt for his valued advice in the preparation of this report; and to Dr. Wm. A. Downes for the privilege of using his records.

STUDIES WITH A NEW METHOD FOR DETERMINING THE COAGULATION TIME OF THE BLOOD IN THE NEW-BORN *

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In pursuance of studies on the coagulation time of blood in the new-born clinic of the pediatric department, University of Minnesota, there was found a host of methods from which to choose, none of which were particularly adapted to the work or gave dependable results.

Since Vierordt's early studies on blood coagulation many ingenious devices and methods have been employed. Hinman and Sladen [1] gave a history and résumé of the work. The more practicable of these methods fall into certain types, and these were studied.

First, was considered Wright's [2] method and its many modifications. These, however, call for the use of special, carefully calibrated tubes with very close temperature control. Even so, results vary widely. Another type of method was introduced by Milian [3] and modified by Duke, Hinman and Sladen.[1] This method, however, gives marked variations according to the quantity of blood deposited on the slide, and Duke's [4] technic requires a specially constructed slide, hard to obtain. Brodie and Russell's [5] instruments, with modification by Boggs, present another scheme. These instruments, however, are special, expensive, require the use of a microscope and are not practicable for general bedside use. Besides, the method employs a current of air directed against the drop. Differences of humidity, temperature and force of the air current introduce factors which affect the results and are hard to control.

Howell criticized all methods employing blood obtained by skin puncture because of the mixture of tissue juices which shorten the

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* Read before the Northwestern Pediatric Society.

1. Hinman, F., and Sladen, F. J.: Measurements of the Coagulation Time of the Blood and Its Application, Johns Hopkins Hosp. Bull. **18**:207, 1907.

2. Wright, A. E.: On a Method of Determining the Condition of Blood Coagulability for Clinical and Experimental Purposes, Brit. M. J. **2**:223, 1893.

3. Milian, G.: Le Causes d'erreur dans l'etude Clinique de la Coagulabilete' du sang, Presse méd. **1**:202, 1904.

4. Duke, W. W.: A Simple Instrument for Determining the Coagulation Time of the Blood, Arch. Int. Med. **9**:258 March) 1912.

5. Brodie, T. G., and Russell, A. E.: The Determination of the Coagulation Time of the Blood, J. Physiol. **21**:403, 1897.

time. His method, as modified by Lee and White,[6] in which the blood is obtained by venipuncture and the clotting time obtained in a small glass tube, is the most often employed. Doubtless, it is the method which, when carefully followed, comes nearest to giving the absolute coagulation time. However, the venipuncture must be done skillfully, with minimal injury to the vessel wall, and the blood must be obtained without the admission of air bubbles, or widely varying results occur. Also, the end result is not sharply defined.

In work on the new-born one is at once confronted with the difficulty of obtaining blood from a superficial vein. It is extremely difficult, often impossible. It is true, one can enter the superior longitudinal sinus, but this requires a special technic. Even the most experienced often fail to enter the sinus because of distortion from molding of the head. It is especially difficult in the first days of life, and besides, the procedure of thrusting a needle through the scalp into the cranial cavity is not always innocuous. It surely is no routine to be practiced by a novice in the home.

A method was, therefore, sought which would require little special apparatus and be simple, easily applied in the home by any physician engaged in the care of the new-born. A return was made to the drop method. All factors such as depth and site of puncture, size and sequence of the drop, temperature, air currents, and the effect of foreign bodies in contact with the blood, were duly considered. An effort was made to control and standardize all these factors and still have the procedure simple and easy of application in the ward or the home. After many trials the following method was evolved.

METHOD

The apparatus required consists of a spring lance (a simple scalpel will suffice), two one and one-half inch watch glasses and No. 6 lead shot, all of which are easily obtained and transported. Glass and shot should be cleaned, preferably by washing with soap and water followed by alcohol and ether. Needless to state, the lance should be sterile, which implies freedom from old blood. The heel of the infant is sponged with ether, a puncture is made with the lance blade set (about 0.5 c.m.) to produce a free flow of blood without the slightest pressure. A clean watch glass containing a No. 6 shot receives the second drop of blood. A second watch glass is inverted over the first. The watch glasses are gently tilted every thirty seconds until the shot no longer rolls, but is fixed in the clot. The end-result is sharply defined, the shot is firmly embedded, so that the glass may be inverted without dislodgment of the shot. At times, due to the forcing out of

6. Lee, R. I., and White, P. D.: A Clinical Study of the Coagulation Time of Blood, Am. J. M. Sc. **145**:495, 1913.

serum, the whole clot may move, carrying the shot with it. This is no source of confusion, for the shot will be found enmeshed in the fibrin and there is no rolling or rotation of the shot. A poorly formed clot or one showing slight retraction is patent, for in such instances the shot continues to roll.

An effort was made to comprehend and check all sources of error. Coating the shot with paraffin did not affect the time. Watch glasses of the same size and curve were employed to standardize the surface area of the drop in contact with glass. The inverted watch glass minimizes the drying of the drop and prevents the entrance of lint and dust. All the work was done in the new-born ward, and the slight variations of the room temperature did not affect results. The greatest source of error is concerned with the blood flow. A small, slowly forming drop obtained by pressure clots very quickly, the tenth drop more quickly than the second. Depth and extent of the cut show no influence as long as the flow of blood is free and occurs without

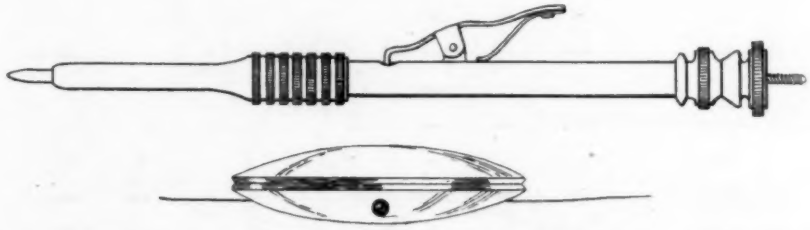

Fig. 1.—The apparatus used consists of a spring lance, two watch crystals and No. 6 lead shot.

pressure or squeezing. In all the determinations the blood flow was free. The first drop was discarded, the second drop, only, employed, and time was reckoned from the moment that the first drop fell. The time consumed in the formation of the second drop was considered a part of the coagulation time. Determinations were made to the nearest half minute.

No claim is made that the absolute clotting time is obtained, but the result is, at least, a clean cut relative time for comparative work. The object is to find gross variations from a normal range. The question is not whether the clot forms in six minutes or in six minutes and forty-five seconds, but whether it forms in six or sixteen minutes.

This method was checked with that of Lee and White in a series of thirty-eight determinations done on the same infants at the same time, blood being obtained for the Lee and White method from the superior longitudinal sinus. The average time was the same—seven minutes—for both methods. This was quite unexpected, for it has been assumed that all methods employing skin puncture give shorter

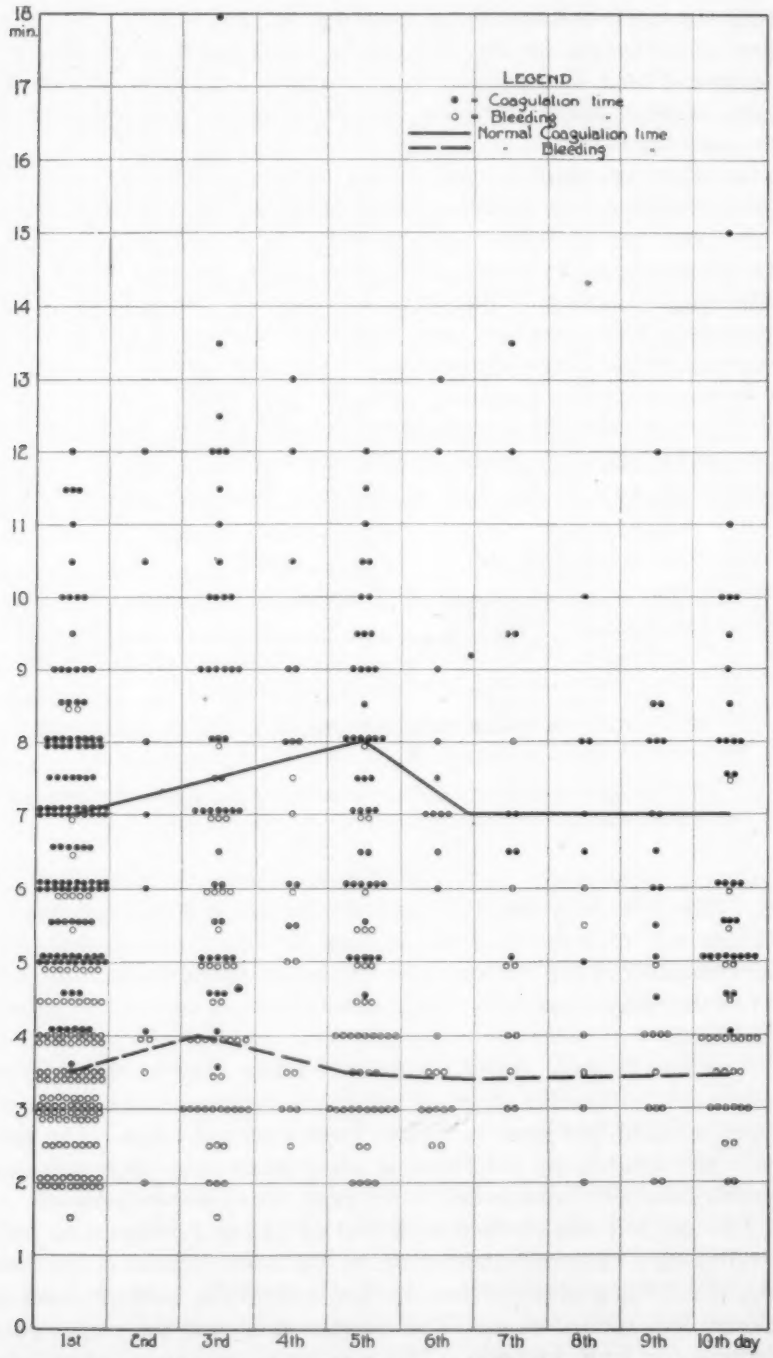

Fig. 2.—Determinations of coagulation and bleeding time on various days of first ten.

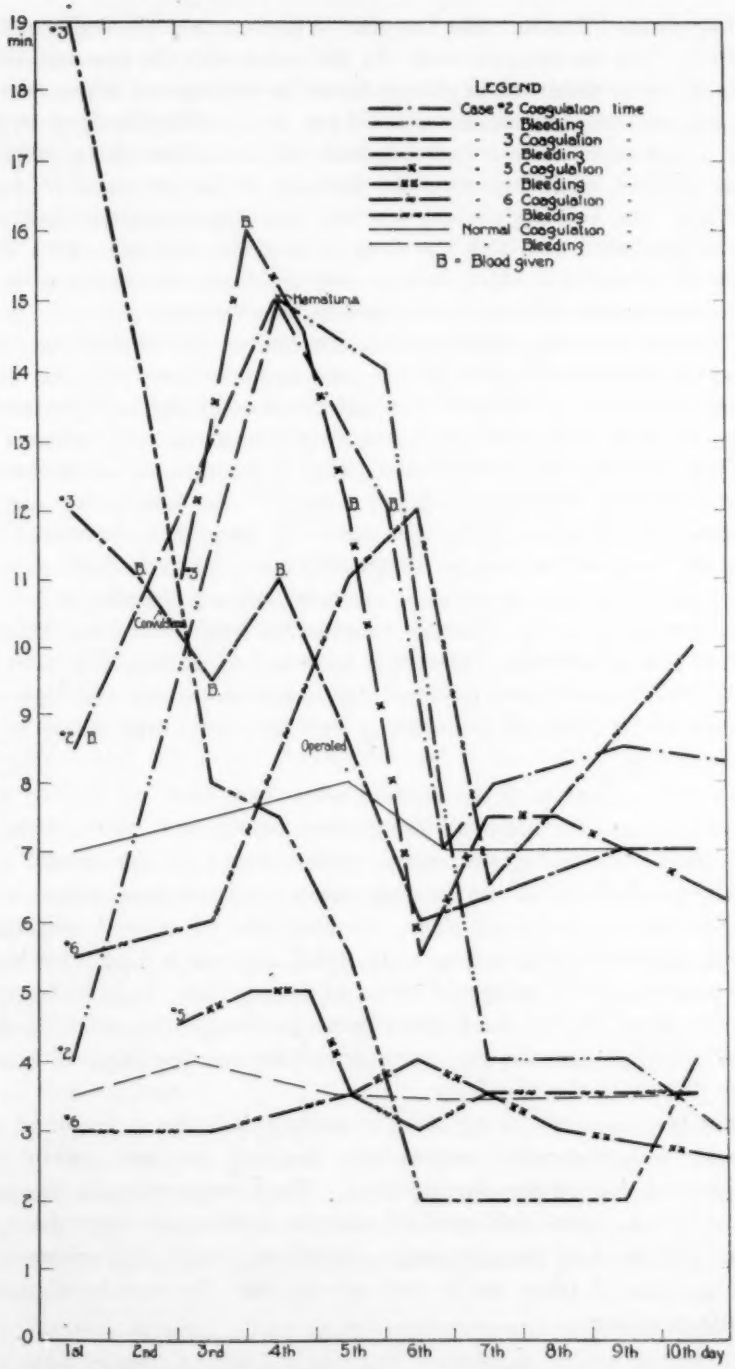

Fig. 3.—Coagulation time in a series of cases above the normal range.

clotting times. Further, the Lee and White method showed a greater variation than the drop method. In the series with the Lee and White method the arithmetical deviation from the average of seven minutes was two and one-half minutes, or 36 per cent., while the drop method gave a variation of one and one-half minutes from the average of seven minutes, or 20 per cent. In the same series the standard deviation with the Lee and White method was approximately three and one-half minutes, and with the drop method two minutes. In a small series of adults and older infants and children the results with the method coincided with those obtained in the new-born.

The average coagulation time determined on 126 new-borns within the first twenty-four hours of life was seven minutes with an arithmetical deviation of one and one-half minutes, giving an approximate range of from five and one-half to eight and one-half minutes, or roughly, from five to nine minutes. Eighty per cent. of all determinations fall within this range, while a range of from four to ten minutes included 95 per cent. of all the cases. It may, then, be stated that with this method the average coagulation time is seven minutes—that a time of more than ten minutes presents delayed coagulation.

It was early noted that the coagulation time varied on different days of the infant's life, showing a tendency to prolongation over the second, third and fourth days with a maximum on the fifth day, and a return to the time obtained during the first twenty-four hours before the tenth day.

Determinations with the method were then made on various days of the first ten, the majority being taken on the first, third, fifth and tenth days. A field graph and a median curve of the results were prepared. Because of the smaller number of determinations on the second, fourth and sixth days, combinations of second and third, fourth and fifth, sixth and seventh, eighth and ninth days were made. The result is then a weighted curve of the median. It is to be noted that the curve reaches the highest points on the fourth and fifth days. It is significant that this corresponds with the age incidence of hemorrhage disease in the new-born.

The bleeding time, using Duke's [7] method, was also determined, and a similar weighted curve constructed. It shows the same general type of curve as that of the clotting time. The average time in the same 126 cases was three and one-half minutes, with an average deviation of one and one-half minutes, and a range from two to five minutes. A bleeding time of from two to five minutes may be considered normal for the new-born.

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7. Duke, W. W.: The Relation of Blood Platelets to Hemorrhagic Disease, J. A. M. A. **55**:1185 (Oct. 1) 1910.

A study was then made of a series of cases presenting coagulation times above the normal range. In this chart the curves of the average normal coagulation and bleeding times are shown, also four abnormal ones with case numbers. It is to be noted, that all these cases presented an exaggeration of the normal curve of coagulation time. It is further to be noted that in two cases a prolonged bleeding time accompanied the delayed coagulation time. In Cases 2 and 3 hemorrhages appeared. In Case 2 a marked hematuria occurred on the fourth day. Blood was given subcutaneously, the reactions returned to normal and the hematuria cleared up. In Case 3 convulsions developed, and other signs of cerebral hemorrhage, on the second day. Blood was given subcutaneously; a decompression operation was performed on the fifth day. Recovery was complete.

On the other hand, cases showing prolonged coagulation time, accompanied by a normal bleeding time, showed no signs of hemorrhage (Cases 5 and 6). It would appear, therefore, that the hemorrhagic factor is a complex of delayed coagulation time and prolonged bleeding time. Further, variations from the normal range of coagulation and bleeding times are due to some deficiency of coagulation properties or excess of anticlotting substances which may gradually be overcome. This may be hastened by the subcutaneous administration of blood (Case 2). In all the cases a return to normal finding occurred by the tenth day. Further studies on calcium, prothrombin, "antithrombin," and fibrinogen content of the new-born blood are under way.

SUMMARY

1. The method is simple, easily applied in the new-born and gave results more accurate than those obtained with the Lee and White method.

2. The average coagulation time in the new-born with the method is seven minutes, with a normal range of from five to nine or ten minutes. The average bleeding time in the new-born with Duke's method is three and one-half minutes with a normal range of from two to five minutes.

3. In the normal new-born there is found a tendency to prolongation of these times in the first days of life. Hemorrhage is accompanied, in many cases at least, with an exaggeration of this tendency or markedly prolonged coagulation and bleeding times.

4. The coagulation and bleeding times should be studied in conjunction.

5. Routine determinations in the new-born will allow the selection of at least certain of the hemorrhagic conditions before the onset of symptoms and give the indication for the administration of blood.

6. The method will allow of a check on blood therapy in hemorrhages of the new-born.

7. Further studies may help to clarify the complex known as hemorrhagic disease of the new-born.

The writer is indebted to Prof. R. E. Scammon of the Department of Anatomy for valuable suggestions and help in preparing the field graphs and curves.

CLINICAL DEPARTMENT

ROENTGEN-RAY DEMONSTRATION OF ABNORMALITIES OF THE GASTRO-INTESTINAL TRACT IN CHILDREN *

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We have thus far made roentgen ray studies of sixty-six cases of chronic gastric intestinal disorders in children from 3 months to 15 years of age. The vast majority were between 3 and 6 years of age. Many roentgenograms were taken, in some instances as many as twenty, in order to determine the emptying time of the stomach or intestine, or both.

CASE 1.—*Megacolon; dilated sigmoid.*—A boy aged 8 years, had an enormous sigmoid pouch with a capacity of at least two pints (Fig. 1). There was displacement of the liver due to the pressure exerted by the massive dilatation of the colon. The bismuth meal did not disappear entirely from the intestinal canal in seventeen days. One can readily appreciate what a difficult matter it would be to carry out any satisfactory treatment in a case of this kind without roentgen-ray examination. There was extreme abdominal distention and moderate malnutrition.

CASE 2.—*Acute pylorospasm.*—This patient was a boy, 7 years of age. The roentgenogram (Fig. 2) shows that the stomach is in active peristalsis, with a pronounced pylorospasm, three hours after the ingestion of the bismuth meal. The boy had been ill for one week with extreme paroxysmal pain in the stomach to such a degree that morphin had to be administered repeatedly. It will be observed that but little of the bismuth meal had passed through the pylorus three hours after the meal was given.

CASE 3.—*Constipation; elongated sigmoid.*—A boy, 6 years of age, came under observation because of obstinate constipation. There had never been an evacuation without assistance since birth. Drugs or local measures were resorted to daily. The roentgenogram (Fig. 3) of the intestine shows that the flexure of the sigmoid passes above the umbilicus and up to the transverse colon, and that the sigmoid is correspondingly elongated.

CASE 4.—*Diarrhea; triple sigmoid.*—This patient was a female, 20 months old, weighing 22¾ pounds. There was a triple sigmoid (Fig. 4). During the early months of her life she suffered from obstinate constipation and about two months before coming under treatment a diarrhea developed which had continued and which it was impossible to control. There was great abdominal distention, assimilation was very defective and death followed from exhaustion. It has been found that in cases of this type a chronic mucous colitis or diarrhea is very apt to follow prolonged constipation.

CASE 5.—*Recurrent vomiting; elongated sigmoid.*—This patient was a girl, aged 4 years. The stomach had not begun to empty in one and one-half hours after the bismuth meal, showing a decided pylorospasm (Figs. 5, 6 and 7). For two years the child had been a sufferer from seizures of recurrent vomiting, the

* Read before the American Pediatric Society, Atlantic City, June, 1919.

attacks lasting from two to four days. It was unusual for three months to pass without an attack. Figure 6 shows that there is retention in the stomach after five and one-half hours. Complete emptying requires about eight hours. Figure 7 shows that the child has an elongated and redundant sigmoid. There was habitual constipation and moderate abdominal distention.

CASE 6.—*Pylorospasm; angulated sigmoid.*—This was a case of marked pylorospasm. The child was a female, aged 3 years, weighing 17 pounds and 3 ounces. She had been subject to periodic vomiting attacks since birth, having a seizure about once every six weeks. There was marked malnutrition, which can readily be appreciated when one takes into consideration her age and weight. She had retention of food six hours after feeding (Fig. 8), which was corroborated by stomach tube examination. In addition to the malnutrition there was a pronounced secondary anemia. The abdominal distention was extreme. Figure 9 shows food residue in the stomach five hours and forty-eight minutes after a bismuth meal. Figure 10 represents the large intestine of the same child. There is a sharp angulation at the junction of the descending colon and sigmoid which probably accounts for the obstinate constipation and the abdominal distention. It was only through the use of enemas or drugs that an evacuation was produced. In angulations of this type it has been found that fecal matter will pass through the intestine more readily than gas.

CASE 7.—*Pylorospasm; wandering stomach; enlarged sigmoid.*—The patient, a female, 5 years of age, had pylorospasm, with the stomach displaced to the left, a so-called wandering stomach. There was a large gastric retention six hours after the bismuth meal (Fig. 11). The retention was repeatedly corroborated by stomach tube examination. The child suffered from habitually poor appetite and there was moderate malnutrition and habitual constipation. The abdomen was greatly distended. Figure 12 shows marked elongation of the sigmoid, extending two inches above the umbilicus and to the right as far as the intestine could pass.

We have found in these patients with elongated and sacculated sigmoids and resulting stasis that the emptying time of the stomach is always delayed. There seems to be direct association between the emptying time of the stomach and that of the large intestine. A peculiar feature of these cases of retention is loss of appetite. The patient will, perhaps, have a fair appetite for breakfast when the stomach is empty. The other meals must be forced or coaxed into the child.

CASE 8.—*Gastroptosis; poor appetite; malnutrition.*—The patient was a female, 16 years of age. There was marked ptosis of the stomach (Fig. 13) and an extreme prolapse of the transverse colon (Fig. 14). This patient was anemic, undersized and underweight. The only active symptom was a persistent pain in the left lower abdominal quadrant. There was no symptom referable to the stomach other than poor appetite and eructation of gas. There was moderate abdominal distention. Medication was required daily to produce an evacuation.

CASE 9.—*Gastroptosis; recurrent vomiting; malnutrition.*—This patient was a male, 11 years of age. The lower portion of the stomach is ptosed 2½ inches below the umbilicus (Fig. 15). The colon is also ptosed to such a degree that with a full bladder, the bladder, stomach and colon would come in close contact (Fig. 16). This boy suffered from periodic vomiting and had been treated for acidosis for several years. There was moderate abdominal distention and habitual constipation. The bowel function required daily supervision.

We have learned a great deal through our roentgen-ray studies. First, we have learned that there may be persistent gastrointestinal derangements affecting the immediate and after-life of the

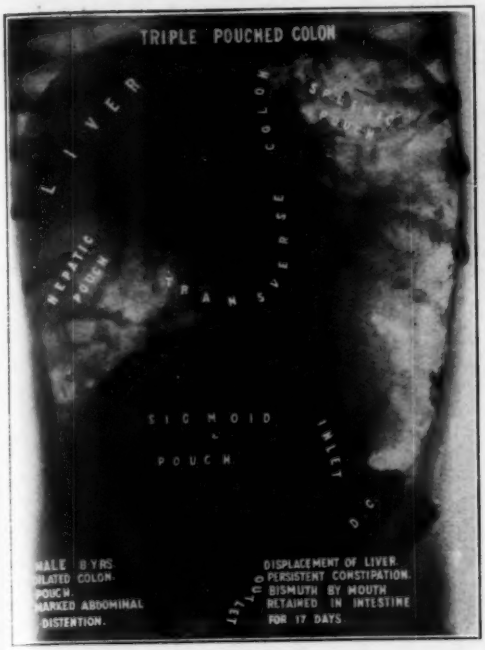

Figure 1

Figure 2

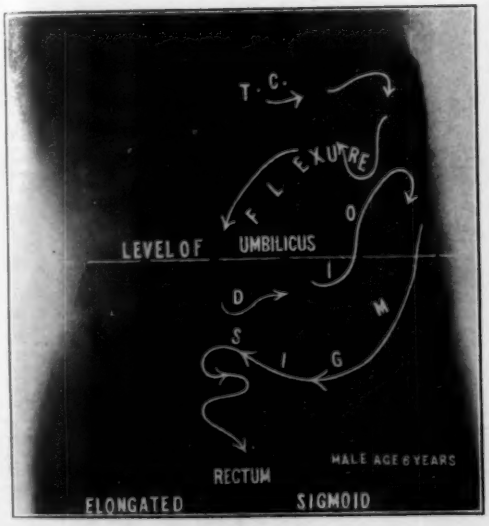

Figure 3

Figure 4

Figure 5

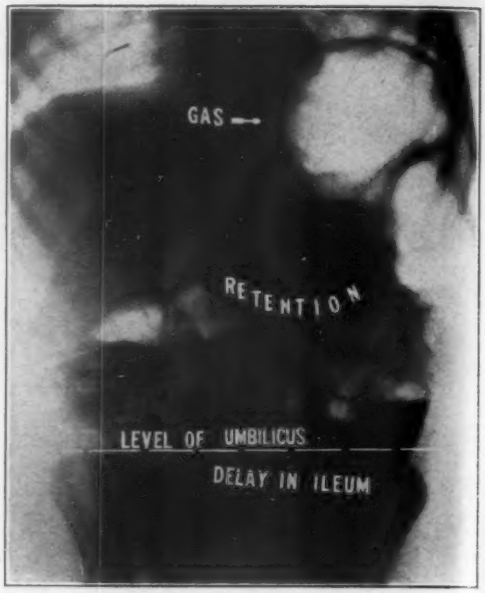

Figure 6

Figure 7

Figure 8

patient, dependent entirely on mechanical agencies. The gastro-intestinal tract of the runabout child has been given the credit of being *structurally* normal, and physicians have relied on examination of the gastric contents, stools and urine with the idea of finding the causes of gastro-intestinal derangements. Our roentgen-ray studies have demonstrated that periodic or so-called recurrent vomiting, which repeatedly had been diagnosed as a manifestation of acidosis, is due, in many instances, to a dilated or ptosed stomach in which there is retention and residue long after the feeding period. Fresh food is given to these children at fairly regular intervals, in a stomach that is never empty, except in the early morning. Nature comes to the relief of the case after a time, and the stomach is emptied periodically at fairly definite intervals.

Another feature of importance relates to the appetite. We have found repeatedly that children who will not eat willingly, and who have to be coaxed and forced, may have a stomach of defective emptying capacity. A child will not be hungry in the event of food remaining in the stomach, beyond the next regular feeding period. It is our effort in these retention cases to leave a long interval between the feedings. Breakfast at 7:30 a. m., dinner at 12:30 or 1 p. m., and supper at 6 p. m. Water only is allowed between meals. With such intervals and measures employed to insure a free daily bowel evacuation, it is surprising how soon many children will develop a normal appetite.

The stomach of the normal child is emptied in from four to four and one-half hours after a meal. Food residue found five hours after a meal means a stomach of slow emptying capacity. We have further proved that there is unquestionably a relation between the emptying time of the intestine and that of the stomach. When intestinal stasis is relieved in a residue case, the stomach empties normally.

In Case 8 there was gastroptosis with surprisingly few active symptoms, other than pain, constipation and capricious appetite. Patients of this type show other symptoms, however, which are of interest. They are always undernourished. They are anemic and irritable in disposition and show faulty growth. They are ill, but the fact is not appreciated because they are as well as usual. They are the type which later in life furnishes work for the abdominal surgeon.

With the view to checking up our observations roentgen-ray studies have been made of children who were not ill. It has been rare to find a dilated or ptosed stomach that did not give rise to symptoms, symptoms which in some instances had not been appreciated. A ptosed colon gives trouble. Not all elongated sigmoids, however, produce symptoms. There may not even be constipation, provided there are no sacculations, adhesions or relaxed abdominal muscles.

Figure 9

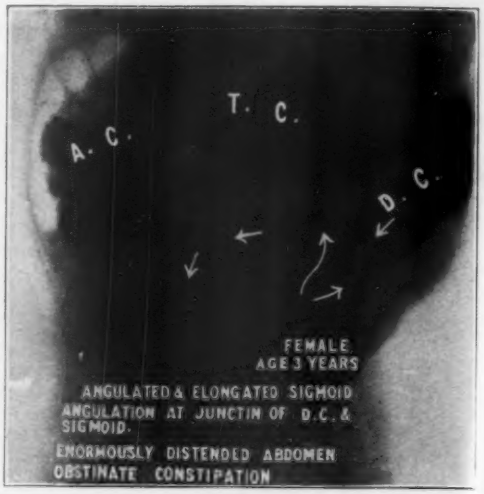

Figure 10

Figure 11

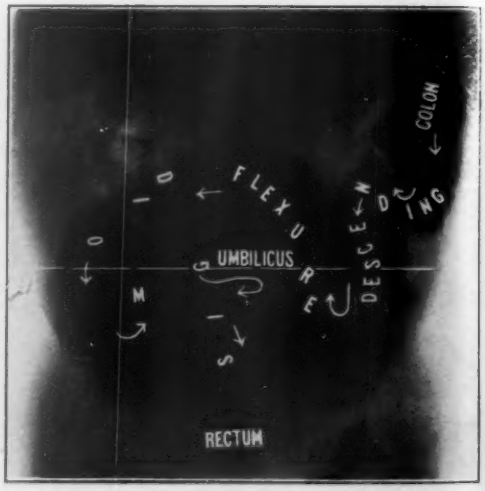

Figure 12

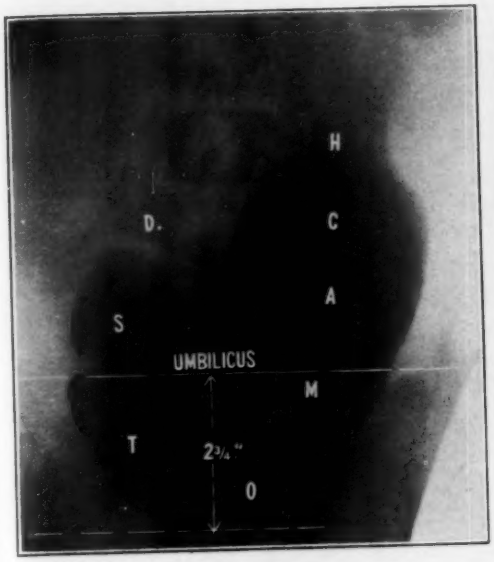

Figure 13

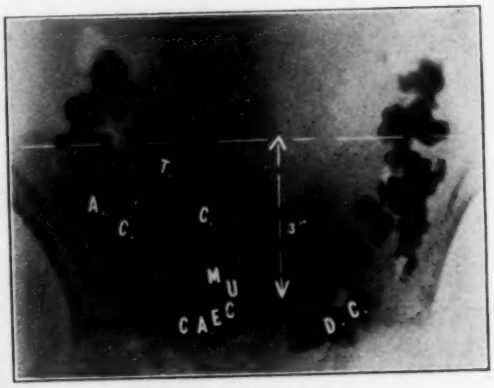

Figure 14

Figure 15

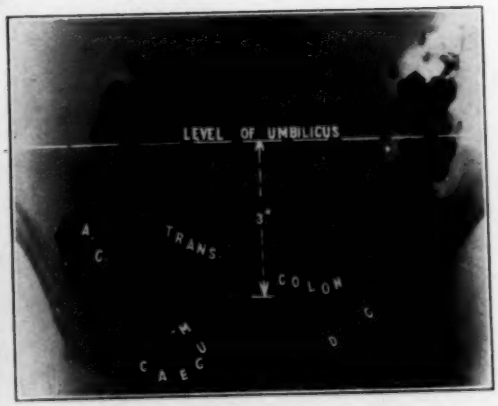

Figure 16

Given a relaxed abdominal wall, a generally distended abdomen and an elongated sigmoid, we have a condition fertile in possibilities for gastro-intestinal disturbances. Weak, relaxed abdominal muscles play an important rôle in these cases. When we supply adequate support, such as a tight-fitting abdominal belt, the relief furnished is very striking. The query has perhaps arisen as to what constitutes a normal sigmoid. I believe that Figure 17 represents such a case. The child was 5 years old. Figure 18 represents a normal sigmoid in a child 8 years of age.

Management.—Only general suggestions as to treatment can be made. Probably the most useful individual measure that applies to

Figure 17

all types of abnormalities, both of the stomach and intestine, is in the use of a well adjusted abdominal belt. For those cases in which the stomach alone is ptosed, a belt with a projecting narrow pad, a so-called shelf, is employed. The device is placed on the belt in such a position as to support the displaced stomach. In those cases in which the intestine alone is involved and in those cases in which there is a gastroptosis, an abdominal belt with a large flat pad is used. The belt is worn constantly when the patient is up and about.

Diet.—In cases of ptosed stomachs in which there is always a delayed emptying, three meals daily only are allowed, and these meals are given at as long an interval as possible. It has been found advisable to give but little fluid with the meals. After each meal, the patient rests in a recumbent position for one hour.

Massage.—In the cases of intestinal stasis, abdominal massage is practiced daily. It has been demonstrated that this measure is of much benefit.

Drugs. — Peristalsis has been shown to be defective when gross intestinal abnormalities exist. In addition to massage for the reestablishing of this function, certain drugs are of value. Small, frequently repeated doses of a combination of nux vomica, belladonna and cascara have been of much aid in producing a more active peristalsis and thus helping to relieve the ever present constipation. But whatever medication is selected, it should be given not less than three times a day. In cases of sacculation and dilatation of the intestine, a full dose of

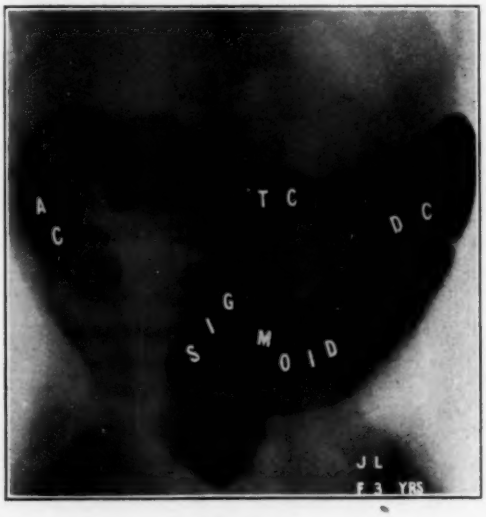

Figure 18

liquid petrolatum is given once a day. Enemas are to be used only as an emergency measure. Atropin is given in full doses when there is an evident pylorospasm.

The roentgen-ray studies on which these observations were made were carried out by Dr. L. T. LeWald, director of the roentgen-ray department at St. Luke's Hospital.

The patient is prepared the evening before by taking a cathartic and the next morning, before the injection, a cleansing soap suds enema is given. A light breakfast is allowed if desired. The opaque substance — barium sulphate — chemically prepared for roentgen-ray purposes, is mixed with fermented milk in the proportion of one part of opaque substance to six parts of the suspending medium. A sufficient quantity is used to fill the colon. This varies with the age of the

patient and the size of the colon, as previously determined by roentgen-ray examination by means of the opaque meal.

The opaque meal is made up of one part of bismuth subcarbonate suspended in eight parts of the ordinary feeding mixture in infants, and for older children it is suspended in fermented milk. If the infant is unable to take the food the ordinary way, it is fed by means of a tube attached to a funnel.

PROGRESS IN PEDIATRICS

RÉSUMÉ OF LITERATURE ON TUBERCULOSIS IN CHILDREN DURING 1918 AND 1919

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MODE OF INFECTION

J. O. Cobb[1] is of the opinion that tuberculous infection in man and animals is generally brought about by the ingestion method. He believes that even where the bacillus is planted on the respiratory mucous membrane by means of dried sputum or by dried feces, as often happens with cattle, the anatomic method is still through the intestinal tract, because the number of bacilli that would be swallowed is out of proportion to the very doubtful number that might possibly reach the air vesicles by inhalation. Cobb agrees with Aufrecht that tubercle bacilli gain entrance into the circulation, however introduced, and then traverse the blood vessels as foreign bodies, until arrested in some organ, most commonly in the pulmonary terminal arteries. Here the bacillus passes through the thin vessel wall barrier, to be picked up by lymphocytes and promptly carried to the nearest lymph gland. Moreover, all portions of the lungs, in man and in animals, are invaded by dust and tubercle bacilli in equal measure, whether the route be through the trachea or through the intestinal tract.

F. Clark[2] says that the importance of tuberculosis can hardly be overestimated, when it is realized that one-fourth of all children give evidence of tuberculous infection. He shows that the route of infection is still of great interest to investigators, and that the majority of pathologists believe that there is always present a primary focus in the lungs. Investigators have also been interested in establishing the fact that tuberculosis in the adult is simply a lighting up of the process which started in childhood. E. L. Opie, however, has shown by a series of necropsies that apical tuberculosis in adults has no relation to the focal tuberculosis occurring in children, nor is it caused by it. Clarke believes that there is not enough evidence for the conclusion that all cases of tuberculosis in adults have their origin in childhood.

Since the publication in 1912 of A. Ghon's work on tuberculosis in childhood, his ideas as to the primary focus in the lung with secondary lesions in the bronchial glands have been generally accepted, as also has been the fact that infection in the adult is only a lighting up

1. Cobb, J. O.: The Point of Election and Modes of Invasion in Pulmonary Tuberculosis, J. A. M. A. **70**:1511 (May 25) 1918.
2. Clarke, F.: Tuberculosis in Children, Nebraska M. J. **3**:158 (Feb.) 1918.

of an old infection of infancy or childhood. Lately, however, the opinion is gaining ground that apical tuberculosis in adults does not depend on an infection of childhood, but is a secondary infection. The work of Opie, quoted by Clark, and abstracted in the résumé of 1917, is strong evidence in favor of this later view.

C. Chomé [3] adds the report of another case of congenital tuberculosis to the few found in medical literature. The child's mother died of tuberculosis twenty-four hours after his birth. On the seventh day the baby developed a cough and an irregular temperature. Auscultation revealed many râles. The child gained weight so slowly that when 2 months old he had not recovered his birth weight, although no digestive disturbance was apparent. The child was always pale, but had red lips. During the second month, tubercle bacilli were obtained from the stools. The endodermal tuberculin reaction was negative. The child died when 3 months old. Necropsy disclosed tubercles scattered through both lungs. The hilus glands were very large and caseated. The spleen showed miliary tubercles. In the tympanic cavities was fluid, from which tubercle bacilli were isolated. Evidently the case was one of congenital tuberculosis. There could hardly be a possibility of infection during delivery, as the child was delivered a few minutes after the bag of waters ruptured. The mother never saw the baby, so that infection later was impossible. The appearance and predominance of the lesions in the bronchial glands leads the author to believe that here the bacilli were first arrested.

BACTERIOLOGY

From her study of the literature, Mildred C. Clough [4] concludes that in all forms of tuberculosis, except the miliary form, the occurrence of tubercle bacilli in the circulating blood in quantities large enough to infect a guinea-pig is relatively infrequent, while in miliary tuberculosis sufficient numbers occur in a large percentage (66.6 per cent.) of the cases. Clough's study reveals only one case in which tubercle bacilli have been cultivated directly from the blood of tuberculous patients. She has, however, been able to grow tubercle bacilli in cultures from the blood of five patients (four of them children) suffering from miliary tuberculosis. In the first three cases she used a medium of 5 per cent. glycerin broth, made neutral or slightly acid. After a preliminary incubation the contents of the flasks were centrifuged and the sediment planted on blood agar slants. In the next two cases the blood was hemolyzed with distilled water and cen-

3. Chomé, E.: Case of Congenital Tuberculosis, Arch. mens. d'obst. et de gynéc. **7**:294 (Oct.-Dec.) 1919.

4. Clough, M. C.: Cultivation of Tubercle Bacilli from the Circulating Blood in Miliary Tuberculosis, Am. Rev. Tuberc. **1**:598 (Dec.) 1917; also Bull. Johns Hopkins Hosp. **28**:363 (Dec.) 1917.

trifuged, and the sediment planted directly on blood agar slants. Clough suggests the use of this method as an aid in the diagnosis of acute miliary tuberculosis, since results are obtained much more quickly than by guinea-pig inoculation.

R. S. Austin [5] analyzed twenty-four cases of tuberculosis in children and infants with special reference to the type of infecting organism. He also made an attempt with each patient coming to necropsy to ascertain the primary focus of infection, basing his conclusions on the lesion which appeared to be the oldest.

Determination of the human or bovine type of bacillus in each case was made by inoculation of rabbits with known amounts of cultures. Each rabbit received also a corneal scratch, into which was rubbed a drop of the bacillus suspension used for inoculating the animal. The other cornea was similarly scratched and rubbed simply with saline solution as a control. The corneal scratch test, however, was not very satisfactory. Although consistently negative in rabbits with the human type of infection, it was not always positive in animals with the bovine type of infection.

Austin found that seven of the twenty-four patients were infected with the bovine type of tubercle bacillus. The primary focus was found in twelve cases. In six cases it was situated in the right lung, in two in the left lung, in three in a bronchial lymph node on the right side, and in the intestine in one case. Although this is too limited a series to permit of drawing definite conclusions concerning the incidence of bovine tuberculosis among children, Austin believes, nevertheless, that the finding of the bovine type of organism in seven out of twenty-four cases emphasizes the etiologic importance of this variety of tubercle bacillus in tuberculosis in children.

R. S. Austin [6] examined for tubercle bacilli the tonsils excised from forty-five children, who clinically showed no evidence of tuberculosis. He made inoculation tests on guinea-pigs, histologic examinations of sections, cultures on Dorset egg medium, and direct smears from the tonsils. In none of the cases did histologic examination, direct smears or cultures show tubercle bacilli. In one instance, a child aged 4 years, the inoculation test yielded the human type of organism. The child's parents were living and well, and there was nothing to point to tuberculosis in the past history of the child. These results tend to confirm the opinion held by many investigators that the surest test for the presence of tubercle bacilli in tissues or other pathologic material is the inoculation test. Austin thinks it of interest that in his one positive

5. Austin, R. S.: Bovine Tuberculosis in Children, Am. J. Dis. Child. **17:** 264 (April) 1919.

6. Austin, R. S.: Bacillus of Tuberculosis in Tonsils of Children Clinically Nontuberculous, Am. J. Dis. Child. **18:**14 (July) 1919.

case the organism should have been the human type of bacillus, and calls attention to the fact that in the few reports on the types of the tubercle bacillus found in the tonsils of children the bovine type predominates. The children in Austin's series, however, came from a community where the supply of cow's milk was not likely to be contaminated with the tubercle bacillus. The author finds that, although tuberculosis of the tonsils in children is not rare, yet, it occurs most often when there are other tuberculous lesions in the body, especially in the cervical lymph glands. The occurrence, however, of the tubercle bacillus in the tonsils of children without clinical evidence of tuberculosis, is not frequent.

SYMPTOMATOLOGY

J. Ritter[7] discusses the course of tuberculous disease at different ages. He says that in infancy and early childhood, if the infection is a massive one, no organ or tissue is spared, and the child dies from a generalized tuberculosis and rarely sees the middle of the second year. Infants, mildly infected during the first year, as well as all children infected during the second and third year, who are either massively or mildly infected, form the great army of children who in the following years are victims of some form of tuberculosis, usually tuberculosis of the tissues and organs supplied by the greater circulation, rather than tuberculosis of the lung. In infancy and early childhood the glands about the hilus and near the bronchial bifurcation, the epibronchial, peribronchial, tracheobronchial, bronchopulmonary and even the tracheal glands are enlarged. These glands usually check the inroads of the virus, and the tuberculous process remains quiescent. Between the ages of 5 and 14 years there is an especial tendency to bone involvement. Up to the sixth and seventh year, the small bones and joints are chiefly affected, but, as the child advances toward puberty, the larger joints, the knee, ankle, shoulder and hip joints are most frequently the seat of tuberculous disease. After the fourteenth year the lungs, which have been more or less spared, are attacked.

The mortality from tuberculosis in early life is 98 per cent. or more; 50 per cent. at the age of 3 years; 20 per cent. at the age of 7, and at puberty only about 2 per cent. After puberty the mortality rate ought to be zero, but at this period many insidious intercurrent conditions arise, which account for the acute, subacute and chronic forms of pulmonary tuberculosis so frequently seen after the fourteenth year. It is interesting to note that the ratio between tuberculosis mortality and infection is in inverse proportion. In early infancy, the infection is usually estimated at between 1 and 2 per cent., with a

7. Ritter, J.: The Course of Tuberculous Disease at Different Ages, Illinois M. J. **32**:416 (Feb.) 1917.

mortality of nearly 100 per cent.; while at the age of 14 years the percentage of those who are infected reaches more than 95, with a mortality of only 2 per cent.

J. P. C. Griffith[8] describes the following types of tuberculosis in childhood:

1. *General Tuberculosis.*—Two clinical forms of this type are seen. (a) The typhoid form: This is sometimes called acute miliary tuberculosis. It is observed chiefly in infancy and in early childhood. The initial symptoms are very vague. There is loss of appetite and weight and general impairment of health, combined with an irregular and entirely uncharacteristic fever. The symptoms strongly suggest typhoid fever, and it is only later, when the time for improvement in typhoid fever should come, that evidences develop of a pneumonia or more often of a meningitis, and the diagnosis of the typhoid form of acute miliary tuberculosis becomes evident. (b) The marantic form: This is observed not infrequently in infancy. The symptoms consist of progressive wasting and anemia. There is no fever, or at most occasional elevation of temperature, and no respiratory or gastro-intestinal disturbances to account for the condition. The symptoms can in no way be distinguished from infantile atrophy due to other causes, and the child finally dies of exhaustion. The diagnosis is made only at the necropsy. Sometimes a few weeks before the end of life evidences of bronchopneumonia develop or other localizing symptoms appear.

2. *Tuberculosis of Special Regions.* — (A) Tuberculosis of the lungs: There are several subdivisions. (a) Acute miliary tuberculosis of the lungs: This is only a form of the general miliary tuberculosis described, in which the localization in the lungs is from the onset the prominent symptom. It is most common about the age of 5 years. There is persistent fever, rapid respiration, prostration, rapid pulse, some cough and sometimes cyanosis. The physical signs are poorly marked. Later, the evidences of tuberculosis elsewhere may show, the child dying possibly from meningitis. At necropsy the lungs are found filled with miliary tubercles.

(b) Acute tuberculous bronchopneumonia: This is a very frequent form of tuberculosis, especially in infancy and early childhood. It may be primary in the lungs, or secondary to tuberculous lesions elsewhere. When bronchopneumonia occurs after measles or pertussis, there is always the likelihood that it may be tuberculous in nature; or when it comes after a period of wasting and ill health. Suggestive, too, is the prolongation of the attack after the time when a simple bronchopneumonia might be expected to resolve.

8. Griffith, J. P. C. Tuberculosis in Children, New York M. J. **109**:485 (March 22) 1919.

(c) Chronic pulmonary tuberculosis: This is the condition which in adult life is called phthisis. It is rarely found in infancy and early childhood and even in later childhood it is uncommon. Another form of chronic tuberculosis of the lungs is subacute or chronic tuberculous bronchopneumonia. This condition is more prevalent. A child may exhibit only moderate, occasional fever, progressive deterioration in health and other vague symptoms, and live for five or six months, only gradually exhibiting the physical signs of bronchopneumonia. Still another form is hilus tuberculosis, in which, in combination with the lesions in the lymph glands, there is a spread of foci of tuberculosis at the hilus of the lungs and along the course of the bronchial tubes. This may develop at any time in early life, even in infancy. The onset is insidious and the symptoms are vague, consisting principally of progressing malaise and debility, with occasional evening rises of temperature and a positive tuberculin reaction. Roentgenographic examination shows involvement of the tissues at the hilus, but does not differentiate the involvement of the lung from that of the bronchial glands.

B. Tuberculosis of the lymph glands: First to be noted in this connection is that of the bronchial lymphatic glands. This is very common, but does not present any positive characteristics. There is usually some loss of health, with more or less fever without discoverable cause. Roentgenographic examination may show decided thickening of the tissues about the hilus of the lungs, caused by disease of the bronchial glands here, the application of d'Espine's sign — the altered voice sound in the neighborhood of the seventh cervical and first dorsal vertebrae — is sometimes useful but cannot be depended on as being absolutely diagnostic. Tuberculosis of the mesenteric glands is a common localization, although less so than localization in the bronchial glands. As a rule, there are no clinically characteristic manifestations. Tuberculous cervical adenitis is by far the most frequent manifestation of tuberculosis in the external glands. It is especially common in early and later childhood. It may be accompanied by other evidences of tuberculosis, but usually is not.

C. Tuberculosis of the alimentary tract: The most frequent form of this variety of tuberculosis is that which is located in the intestine, producing the symptoms of ileocolitis. The diagnosis from other forms of ileocolitis depends chiefly on the discovery of the tubercle bacilli in the stools, or the presence of tuberculosis elsewhere in the body.

D. Tuberculosis of the genito-urinary tract: This is not a frequent manifestation in childhood. Tuberculosis of the testis is seen occasionally, but more rarely than of the female genitals. The kidney is involved in general tuberculosis, and less often the renal structure and the bladder may suffer from a more chronic form of the disease.

E. Tuberculosis of the nervous system: Apart from involvement of the meninges, the nervous system is not often attacked. Large solitary tubercles are sometimes found in the brain and spinal cord, and may give rise to localizing symptoms, but this is uncommon.

F. Tuberculosis of serous membranes: This may occur as one of the manifestations of general tuberculosis or it may predominate in, or be confined to, certain of the serous membranes, especially the meninges, the peritoneum and the pleura.

L. Ribadeau-Dumas [9] describes in detail tuberculosis in infancy. He agrees with most writers that only exceptionally is the disease hereditary. He believes that it is contracted in infancy and its frequency increases with age. The highest points in the mortality curve occur at 8 months and 2 years; but the disease in infancy must not be regarded as always resulting in a general fatal tuberculosis, as was formerly taught. Early pulmonary manifestations do not occur, and failure of general health may be the first sign. The history of contact and result of tuberculin tests are, therefore, very important in infancy. Emaciation is a significant clinical sign, and any infant who fails to gain weight on a suitable diet must be studied carefully for tuberculosis or syphilis. The temperature curve shows, instead of the usual monothermia, a morning or evening rise. A valuable sign after the disease is localized in the bronchial glands is a dry metallic cough, spasmodic, like whooping cough. Sometimes, there is expiratory dyspnea from pressure of the enlarged glands. Auscultation reveals bronchial breathing as low as the fourth or fifth dorsal vertebra. Percussion over the manubrium in front and in the interscapular space behind gives an increased resistance. Pulmonary lesions give few physical signs, and often signs due to enlarged bronchial glands are wrongly interpreted as due to a pulmonary lesion. However, a localized area of râles which is persistent always gives rise to a suspicion of a pulmonary involvement. The author thinks that, on the whole, physical examination of infants is not very satisfactory, but that the roentgen ray can add much valuable information. Frequently, the presence of a surgical tuberculosis or of chronic miliary localization of the disease allows of the correct interpretation of the cachexia, emaciation and anemia. Cutaneous tuberculids, tuberculous gummata and phlyctenular conjunctivitis are manifestations of miliary localization and important aids in diagnosis.

The most frequent form of pulmonary tuberculosis in infancy is the caseous bronchopneumonic form. It shows nothing characteristic, except the failure to resolve and persistence of the physical signs. In

9. Ribadeau-Dumas, L.: Tuberculosis in Children, Progrès méd. **34**:312 (Aug. 9) 1919.

tuberculous pneumonia of infancy there is a marked tendency of the pneumonic areas to break down and form cavities.

Miliary tuberculosis in infancy runs a varied course. As a rule, the greatest number of tubercles are in the lungs and the picture is one of a capillary bronchitis. The roentgenogram is of great help and shows small disseminated irregular shadows, which clear up the diagnosis.

The prognosis of tuberculosis in infancy is grave. Among the author's cases the mortality was from 80 to 85 per cent. The severity of the infection seems to depend on the intensity of inoculation. A massive infection means a rapid course. There is considerable difference of opinion as to whether the cutaneous reaction is a guide to prognosis. Dumas quotes Jeanneret, who believes that tuberculous infants, without marked lesions, react feebly to tuberculin and have a tendency to become immune after repeated injections. Infants with pulmonary lesions are hypersensitive and give a reaction from 30 to 50 mm. in diameter; infants with tuberculous bronchial glands give an average reaction of 14 mm. in diameter. Infants with osseous tuberculosis give a very marked reaction, which remains stationary or diminishes in favorable cases, but increases in unfavorable cases. Infants with meningitis or miliary tuberculosis, at first give a very strong reaction, which becomes more and more feeble as death approaches.

Prophylaxis plays the most important rôle in the treatment of tuberculosis in infancy, and the writer lays great stress on the removal of the child from the tuberculous environment. He thinks the tuberculin, given intracutaneously, as advised by Jeanneret, offers hope in the treatment.

R. Raimondi [10] gives the following symptoms of miliary tuberculosis, which he says often follows an attack of influenza. The infants are very pale and emaciated; early in the disease the appetite is diminished, but later anorexia is very marked. The stools are at first normal, and it is only after the intestines are affected that diarrhea supervenes. Sleep is little disturbed, but the infants cry out during sleep, as if in pain. The inguinal, axillary and submaxillary glands are enlarged. Cough is not a prominent symptom, unless the tracheobronchial glands are much involved. Percussion of the apices, subclavicular regions and infraspinous fossae does not give dulness but a relative resistance. Dulness may be obtained in the region of the hilus. Auscultation of these regions gives an exaggerated puerile respiration. Slight temperature persists through the entire course of the disease, which is from three weeks to three months. The prognosis is always fatal, some of the patients die with meningeal symptoms, some with intestinal symptoms.

10. Raimondi, R.: Tuberculosis in Infants, Presse méd. **26**:593, 1918.

From a careful comparison of a large group of tuberculous and nontuberculous school children, Priestley and Richardson [11] conclude that the weight and height ratio give little or no help in the diagnosis of tuberculosis in children, and that a previous attack of any of the common infectious diseases does not seem to predispose to attacks of tuberculosis. No evidence was found that tuberculosis is more common among those in poor environments than among those in good environment. The figures did not show that enlarged tonsils and adenoids, or caries of the teeth, or heart affections favor the development of tuberculosis. Enlarged cervical glands were much more frequent in tuberculous children than in nontuberculous children, and there were more cases of mental deficiency among them. There were also more instances of eye and ear disease, of anemia and malnutrition in the tuberculous group.

The authors describe the clinical picture of tuberculosis in children attending school. They found 283 cases of tuberculosis of all kinds; in eighty-seven instances the lungs were the seat of the disease. The evidence of pulmonary disorder which was noted in these were: Flattening and retraction of portions of the chest; dilation of the superficial veins; fibrillary contraction of the chest muscles; diminished expansion of part or whole of one or both lungs; diminished movement and increased vocal fremitus; alteration of the normal resonance, from slight impairment to absolute or wooden dulness; diminished respiratory murmur, cogwheel type of breathing, harsh or tubular breathing and adventitious sounds. Not in every case were all or even a great number of these signs present. In some there was little beyond poor expansion, slight dulness, cogwheel breathing and a few crepitations. Cough was present in the majority of the cases, sometimes the short, hacking, dry, spasmodic cough at night, suggesting pressure of enlarged bronchial glands on the recurrent laryngeal nerve. Hemoptysis, pain in the chest, anemia, loss of appetite and loss of weight were also noted.

Among the 283 cases of tuberculosis were 134 cases of glandular disease, involving the cervical glands in all cases, and the axillary glands as well in two cases. Tuberculosis of the glands was found in all degrees of development and severity. In forty-three of the 134 cases abscesses had ruptured spontaneously, leaving typical, bright red scars.

Tuberculosis of the bones and joints occurred in such a great variety of forms and affected so many parts of the body that a composite clinical picture was impossible. The tuberculous process usually affected a single joint or bone, was rarely seen in its active stage, but

11. Priestley, J., and Richardson, B.: Tuberculosis as Seen in Public Elementary Schools, Brit. J. Child. Dis. **15**:183, 1918.

chiefly when quiescent—so that the patients showed the results of the previous disease in deformities of varying degree, atrophy of muscles, marks of old sinuses and ankylosis or stiffness of joints.

Mary H. Williams [12] emphasized two signs in hilus tuberculosis. She says that over the lower part of the chest there is a flattening first on one side, then on the other. Over this area there is also a change in percussion note, which can better be felt than it can be heard. It can also be elicited by direct tapping with the finger tips. The second sign is a bilateral subclavicular flattening and is caused by the downward and backward pull of the fibrous bands connecting the healthy upper lobes with the healing and contracting areas at the base.

From his experience as a pathologist, E. Fisher believes that if chronic hilus tuberculosis does occur in children, it must be very rare. He is of the opinion that chronic disease of the lung in children is almost always nontuberculous, and makes the statement that, if a child acquires a tuberculous infection of the lungs, he is not likely to live more months than he has already lived years. Occasionally, a tuberculous infection may be grafted on a chronic nontuberculous disease. Primary tuberculous infection usually runs an acute or subacute course and very rarely is there found at necropsy evidence of fibrosis.

E. Fisher's statement in regard to the frequency of hilus tuberculosis and its prognosis is not in accord with the observations made by most clinicians. Bronchial gland tuberculosis with hilus involvement is a frequent form of tuberculosis in childhood and very often ends in recovery.

A Krause [14] reports a case of healed multiple tuberculosis in a 14-year-old boy. When 4 years old he developed an osseous tuberculosis following a trauma. This was followed within a year by painless swelling of the epitrochlear and inguinal glands, and by abdominal distention. The lungs were normal at this time. The boy was given tuberculin treatment. The initial dose was 0.00001 mg., and this was increased to 1 mg. The sinus gradually healed and the glands reduced in size. Three years after the onset, however, the child was found to have impaired resonance over the right lung, and enlarged glands could be palpatated in the abdomen. At this time, he was still receiving tuberculin, 0.001 mg. at intervals of two weeks. The next year he developed an axillary abscess, which necessitated extensive dissection of the axillary glands. At the same time, there was a tuberculous

12. Williams, Mary: Hilus Tuberculosis in Children and Adults, Lancet **1**:682 (May 3) 1919.

13. Fisher, E.: Hilus Tuberculosis in Children and Adults, Lancet **1**:814 (June 1) 1919.

14. Krause, A.: Case of Multiple Tuberculosis in Childhood, M. Clinics N. America **2**:1781 (May) 1919.

peritonitis. The patient was then sent to a sanitarium, where he remained seven months. Since then he has not had any recurrence of active tuberculosis.

In discussing the case, Krause emphasizes the element of trauma as often being the exciting cause of an active tuberculosis. He thinks that in this case the primary infection was in the alimentary tract and involved the mesenteric lymph nodes, and from there it spread to other parts of the body. The tuberculin treatment was of little value, but the author believes that the reason for this was that the various foci of tuberculosis were in different stages of development, and it was impossible so to regulate the dosage of tuberculin that all foci would be acted on favorably. The sanitarium treatment was much more satisfactory. The discipline, the regularity of rest, exercise and diet, the life in the open, and the relief from strain, all were factors in producing the cure.

D. B. Leitch[15] reports a case of tuberculous meningitis with xanthochromia and complete coagulation of the spinal fluid in a child 9 months old. Fourteen days before admission, the child had had a little fever and was more restless than usual. Four days later his head was retracted and he was so drowsy that he had to be aroused for his feedings. On admission to the hospital, the patient presented the typical picture of a tuberculous meningitis. A spinal puncture was made, and 15 c.c of fluid were removed. The fluid was slightly turbid and of a light amber color, resembling urine. The cells numbered 250 per c.mm., of which 76 per cent. were lymphocytes. Within ten minutes after withdrawal, the fluid coagulated to a jelly-like mass, so thick that the tube could be inverted without its contents spilling. The patient died on the third day, and postmortem examination showed the typical lesions of a tuberculous meningitis. There was a marked thickening of the membranes in the cervical region of the cord, and the author thinks the adhesions were quite sufficient to cause the obliteration of the subarachnoid space at this level. The spinal fluid in the lower portion of the canal was thus shut off from communication with the fluid in the ventricles, and it is in such conditions that the spinal fluid becomes yellow and coagulates spontaneously on removal.

DeWitt H. Sherman[16] reports an instance of death from tuberculous meningitis in a breast fed infant 10 weeks old. The family history was negative in regard to tuberculosis. The child had never been on the street, had never had any food but breast milk, and no water which had not been boiled. He was taken ill suddenly with convulsions,

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15. Leitch, D. B.: Xanthochromia of Spinal Fluid with Complete Coagulation in Tuberculous Meningitis, Am. J. Dis. Child. **15**:348 (May) 1918.

16. Sherman, H. DeWitt: Death from Tuberculous Meningitis in a Breast-Fed Infant at Ten Weeks of Age, Arch. Pediat. **35**:362 (Sept.) 1918.

developing ptosis of the right eye on the third day. When Sherman saw the child on the third day, the pupil of the right eye was larger than that of the left, and the pupillary reaction of both eyes was sluggish. There was some bulging of the anterior fontanel, and slight rigidity of the neck. The child could be aroused, but made no further response. Tubercle bacilli were found in the spinal fluid and inoculation tests into guina-pigs proved positive. The child died on the seventh day of the illness.

F. van der Boget [17] believes that tuberculous meningitis occasionally, though not frequently, ends in recovery, and reports a case in point. The patient was a child 3½ years old, and, when seen by the author, it had been ill eleven days with a rigidity of the neck which resembled torticollis. The symptoms on which a tuberculous meningitis was based were marked irritability and sensitiveness to external irritation, distinct rigidity of the neck, a temperature irregular in character, reaching 105 F. on several occasions and continuing for fifty-nine days, and the spinal fluid findings, namely, a lymphocyte count of 45 and the presence of tubercle bacilli. A guinea-pig inoculated with the fluid developed typical tubercular lesions. Improvement followed the second lumbar puncture, which was made a month after the onset of the disease, and recovery was uninterrupted. Van der Boget emphasizes the danger of giving the unfavorable prognosis usually given in these cases of tuberculous meningitis; he thinks this influences the family against repeated lumbar punctures, in which lies the only hope of recovery.

From a careful review of the literature, it appears that Van der Boget is correct in regard to the prognosis of tuberculous meningitis. The only patients who have recovered have been those treated by repeated lumbar punctures. V. Bokay collected a series of twenty-nine cases from medical literature, and added the reports of three of his own in which recovery followed this procedure.

According to P. H. Pierson,[18] hemoptysis does not occur in more than 2 or 3 per cent. of tuberculous children. When it does appear as an early symptom, it is usually the result of pressure on and erosion of a blood vessel in the region of the hilus by enlarged or caseous lymph glands. Pierson reports five cases of hemoptysis in a series of 198 cases, seen in the Stanford chest clinic for children. Four of the five children had been exposed to tuberculosis; two were 5 years of age, one was 6 years old and two were 9 years old; four were girls and one was a boy. Physical signs and roentgen-ray findings corre-

17. Van der Boget, F.: Prognosis in Tuberculous Meningitis, Arch. Pediat. **35**:676 (Nov.) 1918.

18. Pierson, P. H.: Hemoptysis in Children, Arch. Pediat. **35**:527 (Sept.) 1918.

sponded very definitely. There was always present increased dulness over the hilus and an increase in harshness in the breathing. Of importance was a musical quality to inspiration and expiration, which denotes an active irritating process. The roentgenograms showed that these signs corresponded to the hilus thickening. Moreover, they showed the extent of the fine markings near the periphery and the dissemination or calcification of the lesions, as they progressed.

E. Bronson [19] reports an instance in which a tuberculous pleural effusion supervened during an attack of erythema nodosum. The child was admitted to the hospital with lesions typical of erythema nodosum. The von Pirquet test was strongly positive. On the eighteenth day there was a rise in temperature, with a serous effusion into the pleural cavity, which later proved to be of a tuberculous nature.

Bronson, discussing the etiology of erythema nodosum, does not believe that it bears any relationship to rheumatism or that it is a specific fever. In this case the strong tuberculin reaction and the clinical association of the two conditions speak in favor of a tuberculous origin, but the comparative rarity of such association in hospital practice speaks against it. The etiology of erythema nodosum seems not yet to have been established.

G. Genoese [20] reviews the literature on chronic hemorrhagic purpura and reports two cases in children 6 and 2 years old, respectively, who soon after the appearance of the purpura developed pulmonary tuberculosis. He believes that the purpura was the first manifestation of the tuberculosis. He quotes Bensaude, who found seven cases of evident tuberculosis and five of probable tuberculosis among thirty-six cases of chronic purpura, and Monnot, who found sixteen cases of tuberculosis among forty-eight cases of purpura. Genoese calls attention also to the fact that giant ecchymoses sometimes appear under the influence of tuberculin treatment.

L. Petitpierre [21] describes a form of acute tuberculous nephritis—which is not a manifestation of acute miliary tuberculosis. It occurs either in the course of a pulmonary tuberculosis or as a primary tuberculosis of the kidney. The onset is usually gradual, with symptoms which point to a disturbance of the gastro-intestinal tract or the onset may be sudden, with pain in the lumbar region, edema of the lower extremities and face, fever and albuminuria. Gastric disturbances are frequent. There is arterial hypotension and a polyuria which is con-

19. Bronson, E.: Erythema Nodosum Associated with Tuberculosis, Brit. J. Child. Dis. **15**:91 (April-June) 1918.

20. Genoese, G.: Chronic Purpura and Tuberculosis, Riv. di clin. pediat. **17**:304, 1919.

21. Petitpierre, L.: Acute Tuberculous Nephritis, Arch. de med. d. enf. **21**:467 (Sept.) 1918.

stant and of diagnostic import. Hematuria is persistent. A family history of tuberculosis is helpful in establishing the diagnosis, but it must be confirmed by finding the tubercle bacilli in the urinary sediment through staining methods or animal inoculation. A focal reaction after an injection of tuberculin is also confirmatory. The author's case is as follows: an 8 year old child was admitted to the hospital for acute hemorrhagic nephritis. Five months before albumin had been found in the urine and just before entrance albumin and blood were found. The mother was suffering from pulmonary tuberculosis. Examination showed a somewhat emaciated child, with impaired resonance and increased vocal fremitus over the right pulmonary apex. The heart was enlarged to the right and there was accentuation of the second aortic tone. The urine contained albumin and blood. The von Pirquet, Mantoux and Wassermann tests were negative. The albumin gradually disappeared but the blood persisted. Diuresis increased progressively, until the child was passing from 800 to 1,000 c c. of urine in twenty-four hours. After three weeks in the hospital, there was an acute exacerbation. The albumin soon disappeared again, but the blood persisted. Two months after entrance, the child developed a phlyctenular keratoconjunctivitis. A diagnosis of tuberculous nephritis was based on the successive attacks of hematuria, the polyuria, the arterial hypotension, the finding of a pulmonary lesion, and the appearance of the phlyctenular keratoconjunctivitis. A year after entrance to the hospital, the urine was normal. The child was given 3 mg. of tuberculin, when albumin and blood at once appeared in the urine. The author's observations show that in considering the etiology of nephritis, tuberculosis must be kept in mind. Also the likelihood of recurrences in this form of nephritis must not be forgotten.

Petitpierre's report is of especial interest, as tuberculosis is not commonly considered a cause of hemorrhagic nephritis. This possibility may clear up the origin of an occasional obscure case.

J. C. M. Fournier [22] reports an instance of Raynaud's syndrome in a tuberculous child. The patient, a boy, aged 12 years, was admitted to the hospital with evidence of tuberculous involvement of the right apex. Three weeks later there were well marked signs of meningitis. At the same time he developed a symmetrical asphyxia of the upper and lower extremities. Both hands and feet were cyanotic, cold, and painful. This condition gradually subsided but recurred to such a marked degree that gangrene of the limbs was feared. It again subsided, to recur still another time. The child died seven weeks after admission. Necropsy showed tuberculous meningitis, pleurisy, caseous areas in the lungs and caseous lymph nodes.

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22. Fournier, J. C. M.: Raynaud's Syndrome in a Patient with Tuberculous Meningitis, Abstr., Arch. de méd. d. enf. **22**:438, 1919.

W. Stanley Gibson [23] has made a careful study of ninety-two cases of phlyctenular conjunctivitis. The investigation included an exhaustive history, a painstaking physical examination, and various laboratory tests, with special attention to the numerous views as to the cause of this condition which have been brought forward. Among these views the following were considered sufficiently important to merit separate discussion: (1) impairment of the general health; (2) bad living conditions; (3) gastro-intestinal disturbances; (4) the exudative diatheses; (5) tuberculosis.

Patients with phlyctenular conjunctivitis did not differ in regard to general health and development and the history of previous illnesses from those presented for other complaints. Bad living conditions were not frequently found, and the author thinks that they play a factor much less important in the production of this condition than is generally imagined. As to gastro-intestinal disturbances, the patients seemed particularly free from complaints referable to the gastro-intestinal tract. There were several typical examples of the exudative diathesis, and sometimes the presence of tuberculous cervical glands completed the picture of scrofula. But even to assume that phlyctenular conjunctivitis is a manifestation of the exudative diathesis would amount only to a restatement of the problem, for the cause of the so-called diathesis is altogether obscure.

The frequent occurrence of phlyctenular disease not only in scrofulous children, but also in those who showed other clinical types of tuberculosis suggested the possibility of the conjunctivitis resting on a tuberculous basis. A von Pirquet test was positive in all but two cases, and these were the only two cases in the series not typical of the disease. The author believes that the tuberculous theory of the etiology of phlyctenular conjunctivitis brings into harmony the other apparently conflicting opinions as to its cause. Impairment of the general health, bad living conditions, and insufficient food furnish favorable opportunity for tuberculosis to gain a foothold. Gastro-intestinal disturbance may be a symptom of obscure tuberculous infection. The exudative diathesis gives a soil favorable to the entrance and growth of the tubercle bacilli, which may then produce the clinical picture known as scrofula.

The author's experiments on rabbits also point to a tubercular origin of phlyctenular conjunctivitis. Phlyctenules were produced experimentally in tuberculous rabbits in eight instances, six times as a complication of conjunctival reactions resulting from the instillation of tuberculin, twice in the absence of local irritation of any kind. Numerous attempts to produce the condition in nontuberculous rabbits were unsuccessful. The pathology of the experimental lesions was similar

23. Gibson, W. Stanley: Etiology of Phlyctenular Conjunctivitis, Am. J. Dis. Child. **15**:81 (Feb.) 1918.

to that of phlyctenules in human beings. The microscopic findings suggest a tuberculous origin. The author concludes that all clinical, experimental and pathologic evidence points to tuberculosis and tuberculosis alone as the cause of phlyctenular disease.

C. Combeléran [24] reports a case of tuberculosis of the vulva in a child 1 year old, whose mother had tuberculosis. According to the history given by the mother, the lesion on the vulva was preceded by swelling and the discharge of pus from the right inguinal glands. On examining the child, the author found an ulcer of a week's duration, which involved the right labium minorum and the right side of the fourchet. The base was of a pale rose color, with projecting whitish points about the size of pin points. In the right inguinal region was a broken down gland, and in the left inguinal region were two enlarged lymph glands. There were no physical signs in the lungs, but the child developed a cough and tubercle bacilli were found in the sputum. Microscopic examination of the tissue from the ulcer showed tuberculous changes. The ulcer was excised and curetted, but six months after the onset was still not healed.

The author collected thirty-seven cases of tuberculosis of the vulva from medical literature, and found that in most instances they were of primary origin. The diagnosis is sometimes difficult and often necessitates histologic examination.

F. Tits [25] describes tuberculosis of the middle ear. He says that infection rarely takes place through the drum membrane, that it usually travels from the nasal fossae or from the pharynx through the eustachian tube. Occasionally, in patients suffering from osseous tuberculosis it is carried through the blood stream. The chief form in children is the so-called necrotic form. It occurs in children who have not a severe tuberculous disease. It is characterized by an abundant discharge of fetid pus and early facial paralysis. The patients frequently complain of noises in the affected ear and of vertigo. Symptoms of acute mastoiditis usually occur, and death results from a meningitis. At other times, the disease runs a much slower course, with necrosis of the internal ear and fistula of the mastoid. The diagnosis is based on the general condition of the patient, the presence of visceral or osseous tuberculosis, the presence of multiple perforations of the drum, the resistance to all treatment, and the finding of tubercle bacilli in the aural discharge.

The possibility that a chronic discharge from the ear is of tuberculous origin should always be kept in mind by the pediatrician. It may lead to a diagnosis and the institution of proper treatment before the process has become too widespread to be influenced.

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24. Combeléran, C.: Tuberculosis of the Vulva, Internat. Clin. **2**:158, 1918.
25. Tits, F.: Tuberculous Otitis Media, Arch. méd. Belges **71**:168 (Aug.) 1918.

TUBERCULOSIS OF THE TRACHEOBRONCHIAL AND CERVICAL LYMPH GLANDS

L. J. Moorman [26] discusses tuberculosis in children, with special reference to tracheobronchial gland tuberculosis. He quotes Krause, whose recent experiments, he thinks, prove that the tracheobronchial glands may ultimately receive tubercle bacilli which have traveled from remote parts of the body and failed to find any demonstrable lodgment in the lungs. He does not agree with Ghon that all cases are air borne, with a primary focus in the lung and a secondary focus in the bronchial glands.

Moorman emphasizes the fact that the great majority of children are infected, and that the infection may at any time become a clinical tuberculosis. He asserts, too, that the first symptoms are those of a toxemia, namely, weakness, undue fatigue, poor appetite, failure to gain in weight and nervous irritability. This group of symptoms becomes particularly significant when there is a history of contact with tuberculous individuals, or when there are signs of local involvement, such as cough and hoarseness. In these instances a thorough examination of the chest should be made, with special reference to interscapular dulness and the presence of d'Espines sign. Diminution in the intensity of breath sounds on the side of greatest involvement is suggestive, and is probably due to pressure from enlargement of the hilus group of glands. The roentgen-ray examination is important, but the results must be interpreted in the light of the history and physical examination. The von Pirquet test is of value in diagnosis but must be repeated two or three times before it can be considered as being negative.

Moorman examined 135 children at the Oklahoma City Tuberculosis Dispensary. Of these, fifty gave a history of exposure to open cases, seventy were exposed to suspected cases and fifteen gave no history of exposure. A history of the following symptoms was obtained: cough in 59 cases; expectoration in 29; pain in chest in 24; pleurisy in 14; hemoptysis in 2; night sweats in 11; loss of flesh in 25; loss of strength in 34; hoarseness in 17; fever in 19; loss of appetite in 28; disturbed digestion in 27. Physical examination revealed the following: weight below standard, 73; height below standard, 17; general appearance good in 95, fair in 48, poor in 21. Type of chest normal in 121, abnormal in 14, dulness elicited by percussion in 89; d'Espine's sign was present in 35; râles in 29. Sixty-nine children were examined roentgenographically and the roentgenogram showed tuberculosis of the tracheobronchial glands alone in 11 cases; tuberculosis of the tracheobronchial glands with peribronchial thickening in 29 cases; tuberculosis

26. Moorman, L. J.: Tuberculosis in Children with Special Reference to Tracheo-Bronchial Gland Tuberculosis, Oklahoma M. A. J. **12**:123 (May) 1919.

of the tracheobronchial glands and peribronchial thickening with involvement of the lung tissue in 19 cases; nontuberculous infection in 3 cases. The diagnosis as recorded in the 135 cases was: tuberculosis of the hilus alone in 34 cases; tuberculosis of the hilus with peribronchial and lung involvement in 44 cases; doubtful, 40 cases; cases with nontuberculous infection, 5; negative, 12 cases.

J. C. McGowen[27] describes the clinical and roentgen-ray diagnosis of bronchial gland tuberculosis. He says that loss of weight, combined with a dry, hacking cough, paroxysmal in character, with frequent recurrences at night, are suspicious symptoms. Attacks of pain in the region of the heart without apparent cause, occasionally accompanied by a fit of coughing, are produced by compression of the vagus nerve by enlarged bronchial glands.

Enlargement of the glands of the neck and supraclavicular spaces are often associated with bronchial gland infection, as are also enlargement and swelling of the superficial veins of the chest. Dulness in the oval space on each side of the second, third and fifth dorsal vertebrae is significant, but is not caused by the glands themselves but by interference with lines of communication to the surface structure by the glands. Dulness over one or both apices and in the oval space, with localized and persistent fine crepitant râles, is almost conclusive evidence of the presence of active tuberculous disease.

McGowen thinks that roentgen-ray examination gives more valuable evidence as to the presence of tuberculosis than physical examination, but it gives no proof of the activity of the disease.

In early tuberculous disease in children, when either the bronchial glands or the lung tissue are involved, there is an increase in the normal hilus shadows, especially on the right side. The trabeculae of the lung are thickened, and, if the disease has progressed to any extent, irregular, circular patches are seen in the pulmonary parenchyma.

E. Schmieglow[28] remarks that perforation of tuberculous glands into the air passages or the esophagus has often escaped detection during life, and necropsies have shown that this was a cause of death from suffocation. He says that it is possible by modern methods to determine whether enlarged bronchial glands are responsible for stenosis of the air passages and to anticipate this catastrophe. Schmieglow tabulates twenty cases of perforation of the bronchial glands into the bronchi which he collected from the literature, and reports a number which have come under his own observation. He says that symptoms of compres-

27. McGowen, J. C.: Early Diagnosis of Tuberculosis in Children, Brit. J. Tuberc. **12**:145 (Oct.) 1918.

28. Schmieglow, E.: Tuberculous Bronchial Gland, Hospitalstid. **61**:129 (Jan. 30); 161 (Feb. 6) 1918; Abstr., Am. Rev. Tuberc. **2**:581, 1918.

sion by enlarged glands are: expiratory dyspnea, a cough which resembles whooping cough and occurs spasmodically, an area of dulness in the interscapular region and a peculiar whistling character of the respiration. When suppuration and perforation into the bronchus occurs, the child may cough up the softened gland, but often it is too large, and suffocation results if operative procedures are not resorted to. Symptoms of stenosis are an indication for bronchoscopy and it may be possible to aspirate the contents of the gland through the bronchoscope. When the enlarged glands are in the anterior mediastinum, they can only be reached by removal of the sternum. Tracheoscopy is especially useful in cases which develop gradually. In children under 6 years of age, it is necessary to perform a tracheotomy before tracheoscopy, but not in older children.

The following standards[29] of diagnosis and treatment of tuberculous cervical adenitis have been prepared by the National Tuberculosis Association: Enlarged cervical glands in children under 2 years of age are usually due to some acute inflammatory process rather than to tuberculosis. Any well marked enlargement of the anterior cervical glands, that persists for several months after throat and mouth infections have been relieved, should be considered tuberculous. Tuberculosis should be considered seriously as a cause of enlarged cervical glands when there has been known exposure to tuberculosis from human or bovine sources. Enlargement of cervical glands may take place following or during acute infectious diseases of childhood, acute or chronic tonsil infections, pharyngitis, carious teeth, leukemia, Hodgkin's disease, syphilis, middle ear disease, eczema, mycosis or irritation of the scalp. Enlargement of the glands posterior to the sternomastoid are usually nontuberculous. Tuberculosis should be considered seriously if constitutional signs and symptoms, such as anemia, malnutrition, fever, rapid pulse, loss of weight and strength, accompany enlarged cervical glands. Tuberculosis should not be considered the cause, unless the enlargement has persisted for several months, but it is a safe rule, after having to discard other diagnosis, to regard as tuberculous any gland which has been enlarged for three months or more. In the presence of evident focal infections, as diseased tonsils or adenoids or carious teeth, it is better to take it for granted that such glands are not tuberculous, until the effects of the removal of the tonsils and adenoids and the clearing up of the teeth has been observed. A pathologic examination of a portion of gland should be made if there is any doubt as to the diagnosis, and the removed tonsil should

29. Standards of Diagnosis and Treatment of Tuberculous Cervical Adenitis, Am. Rev. Tuberc. **2**:565, 1918.

be examined for tuberculosis histologically and by animal inoculation. Confirmatory evidence of the tuberculous nature of the enlarged glands may be secured through the tuberculin test and the roentgen ray, and in manifest tuberculous conditions in other parts of the body.

Tuberculous cervical adenitis should always be treated as a general disease with local manifestations. All possible portals of entry of infection, such as teeth, nasopharynx, tonsils, ears and scalp should be examined and all infections treated. All methods of treatment should be considered carefully in each individual case, and whatever line of treatment is instituted, hygiene should play a most important part. Tuberculin, surgery, heliotherapy, and the roentgen ray may be valuable in the treatment of tuberculous cervical adenitis, but only when associated with hygienic treatment.

R. S. Haynes [30] says that an adenitis in a child under 2 years of age is likely to be a simple rather than a tuberculous inflammation; when the child is over 2 years of age, it is more likely to be tuberculous; but widespread tuberculous adenitis may occur in susceptible infants. A knowledge of the diet, of the milk used and of the child's surroundings, is important in diagnosis. A negative history of contact is more important than a positive one. According to Haynes, tuberculous adenitis is rarely acute. A slow increase of the swelling, with gradual involvement of more than one node, and delayed involvement of the skin and deeper structures speaks for tuberculosis. In early infancy, however, a rapid and acute onset may characterize tuberculous swelling and cause difficulty in diagnosis. When an enlargement has lasted more than three months, the process is probably tuberculous, although in infancy mild simple infections may run such a course. The presence of a single, slowly enlarging node, particularly if it is a tonsillar node, points to tuberculosis. Simple adenitis usually presents a fairly early stage enlargement of several nodes; tuberculous adenitis shows in a late stage enlargement of several nodes. Inflammation of the structures in the mouth, throat and nasopharynx points to the diagnosis of simple or pyogenic, rather than tuberculous enlargement of a node. A persistent discharging sinus points to a tuberculous infection. Examination of the blood shows a polynucleosis in the pyogenic enlargements and a lymphocytosis in the tuberculous enlargements, but secondary infection in tuberculous adenitis also causes a polymorphonuclear leukocytosis. A negative von Pirquet test is valuable evidence of a pyogenic infection. The author emphasizes the difficulty of diagnosis and frequently finds it necessary to excise and examine a gland before definitely determining it.

30. Haynes, R. S.: Diagnosis of Enlarged Lymph Nodes, Arch. Pediat. **35**:226 (April) 1918.

DIAGNOSIS

M. Fishberg [31] criticises the standards of diagnosis of pulmonary tuberculosis in children issued by the National Association for the Study and Prevention of Tuberculosis. In the first place, he does not think that such a polymorphous pathologic process as tuberculosis can be standardized. He points out that malnutrition, on which much emphasis is laid, may be caused by other factors than tuberculosis, that pleurisy is not always of tuberculous origin in children; that lassitude and fretfulness, symptoms attributed to tuberculosis, are the usual symptoms of a lymphatic diathesis; that night sweats are seen in physically healthy children and are significant only when they occur in the early morning hours. He points out that an elevation of temperature in children is not a positive indication of a serious process. With regard to physical signs, he takes issue with the statement that râles occur infrequently in the apices and along the border of the sternum. In cases of pulmonary tuberculosis in children, Fishberg has always found râles in the apices. He emphasizes that the time has passed when a positive tuberculin reaction was sufficient as a basis for a diagnosis of tuberculous disease, even in a child with some of the above symptoms and signs. In regard to the statement that pulmonary tuberculosis should be suspected in patients with glandular, osseous and articular tuberculosis, Fishberg states that pulmonary tuberculosis is exceedingly rare in individuals of any age who are suffering from these forms of the disease. He takes exception also to the statements that on an unexplained hemoptysis, with a positive tuberculin reaction, with or without a definite positive roentgenogram, a diagnosis of pulmonary tuberculosis is to be made, and given a roentgenogram which shows unmistakable mottling, with a positive tuberculin reaction in a child under 3 years of age, even if constitutional symptoms are absent, the diagnosis of pulmonary tuberculosis is to be made. He thinks that expectoration of blood comes usually from enlarged tonsils and adenoids, and that a roentgenogram of a child over 4 years of age, which shows no mottling or opacities in the region of the hilus, is very rare. Fishberg has seldom encountered pulmonary tuberculosis in children between 4 and 12 years of age, but he has seen many cases of tracheobronchial adenopathy in children, and is surprised that this form of tuberculosis is not even touched on in the standards laid down by the committee.

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31. Fishberg, M.: A Criticism of the Standards of Diagnosis of Pulmonary Tuberculosis in Children Issued by the National Association for the Study and Prevention of Tuberculosis, New York M. J. **106**:967 (Nov. 24) 1917.

J. Hess [32] discusses the various tuberculin skin reactions in children. These are divided into qualitative and quantitative tests. The simplest of the qualitative tests is the von Pirquet. Hess describes six types of reaction following the von Pirquet test: (1) The usual typical tuberculin reaction, which begins within four or six hours, reaches its maximum in from twenty-four to thirty-six hours, and then rapidly fades, although infiltration persists for several days. (2) A premature reaction, characterized by rapid evolution and slight intensity. (3) A persistent reaction, which reaches its maximum slowly and then remains unchanged for a week or longer. (4) A late reaction, which makes its appearance after an incubation period of more than twenty-five hours. (5) A cachetic reaction, characterized by infiltration with little or no redness. (6) A scrofulous reaction in which there are one or more papules, surrounded by a painful area of redness from 5 to 10 cm. in diameter.

Quantitative tests, according to Hess, are of little practical value. The intercutaneous test is the one most extensively used. Hess makes four simultaneous injections, (a) 1/20 c.c. of salt solution as a control, (b) 1/20 c.c. of a 1 : 1,000,000 solution of tuberculin, which equals 0.00005 mg.; (c) 1/20 c.c. of a 1 : 100,000 solution, or 0.0005 mg.; (d) 1/20 c.c. of a 1 : 10,000 solution, or 0.005 mg. If there is no reaction, he uses 1/20 c.c. of a 1 : 1,000 dilution, or 0.05 mg. and 1/20 of a 1 : 100 dilution, or 0.5 mg. When the test is positive, a pink infiltration appears within several hours, increases during the first twenty-four hours, and attains its maximum on the second day. This test has four advantages over the von Pirquet, the more exact dosage, the earlier reaction, the more distinct reaction and its greater reliability, because of certainty of absorption of the tuberculin. The subcutaneous and percutaneous tests are less extensively used. Hess agrees that the tuberculin skin reaction is a specific reaction. It gives indication of present or previous tuberculous infection, but not necessarily of active tuberculous disease. The quality of the reaction is of little importance for the diagnosis of the intensity of the process. A negative reaction to the von Pirquet cutaneous tuberculin test, especially if made twice, occurs only in individuals entirely free of tuberculosis or in individuals suffering from acute infectious disease, during pregnancy, after introduction of large doses of tuberculin, in cachetic conditions and in advanced and miliary tuberculosis. Hess believes that the tuberculin reactions should be considered as adjuncts to careful clinical examination and observation, and not as a definite means of diagnosis or as establishing a prognosis.

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32. Hess, J. H.: Tuberculin Skin Reactions in the Diagnosis of Tuberculosis in Children, Med. Clinics N. America **1**:1357, 1918.

E. F. Warner [33] has selected the subcutaneous, the von Pirquet, the Mantoux, and the Ellis papillary cutaneous tuberculin tests as the most practical for use in children. He thinks that the subcutaneous is one of the most valuable, and that it is too much neglected by pediatricians, on account of the time it involves and the unfounded fear of violent reactions. A positive von Pirquet simply implies that the child has at some time become infected with tubercle bacilli. The greatest value of the intradermic test is to determine the degree of hypersensitiveness of patients with active tuberculosis, in order to regulate the dose of tuberculin with which to commence treatment.

Warner believes that the Ellis multiple papillary cutaneous test is the most valuable for separating the active from the latent cases. The test is performed as follows: Scarifications are made with an ordinary vaccination lancet through each of seven drops of solutions of tuberculin placed in a row on the inner side of the forearm. Scarifications must open the papillary layer of the skin, to draw blood. The solutions used are bovine tuberculin, full strength, for mark 1. The other five solutions are made from old tuberculin of the human type. For mark 2 a 1:10 solution is used; for mark 3, a 1:100 solution is used; for mark 4, a 1:500 solution is used; for mark 5, a 1:1,000 solution is used; for mark 6, a 1:10,000 solution is used; mark 7 is used as a control. A high colored, edematous reaction denotes an active lesion, a dark colored reaction denotes an old or quiescent lesion, if it is nearly black, with no swelling, the patient's condition is hopeless. No reaction is shown in a far advanced case. Incipient cases yield a reaction to mark 1 or 2. Probably, if there was a reaction to mark 6, the amount of lung tissue involved in the tuberculous process would be a very small patch, just definitely detectable by the stethoscope. The usual moderate lesions give reactions on mark 3 or 4. A reaction on mark 3 is the most important for the pediatrician, as it is the dividing line between active and latent lesions.

In twenty-three Ellis tests, Warner obtained ten positive reactions to mark 4, three to mark 3 and ten negative reactions. The results in this small series show only three active cases out of twenty-three, or a little more than 10 per cent. The author thinks further investigations by means of a reliable tuberculin test should be undertaken to separate active from latent cases of tuberculosis.

There seems to be considerable difference of opinion in regard to the relation of the severity of the cutaneous tuberculin reaction to the activity of the tuberculous process. Most writers believe little can be

33. Warner, E. F.: A Comparison of the Various Tuberculin Tests in Childhood, Arch. Pediat. **35**:28 (Jan.) 1918.

told from the type of the reaction. Perhaps Warner's report will stimulate more investigations along these lines and his conclusions will be proved to be accurate.

E. M. Sill [34] subjected 658 children, ranging in age from 2 months to 13 years, to the von Pirquet test. The children were the average type of the lowest East side of New York. The parents were mostly of foreign birth, but in no case was there a family history of tuberculosis. A positive reaction was found in sixty-one or 9.2 per cent., figures, which seem to give a fair estimate of the frequency of infection with tuberculosis in children with healthy parents in New York City. Sill thinks that in young children a positive reaction indicates a recent infection that is active or partially healed, while in older children the intensity of the reaction indicates the hypersensitiveness of the child, rather than the degree of the disease. When the reaction comes on quickly, it means an active focus; a slow or delayed reaction means a latent focus. Some children do not react to the first dose, but they do react to the second test. Comparing the results of the present investigations with others, the author concludes that the incidence of tuerculosis infection in children varies not only in different countries but in different states of the United States, and varies also among children in hospital and clinic practice.

M. H. Bass [35] is of the opinion that, in spite of the advance in knowledge, there is still a great deal to be learned about the tuberculin reaction as it affects children. He was led to collect data on this subject by the fact that he found in an asylum a number of children with negative reactions, who had reacted positively six months before. In many of the children the test was repeated several times in the course of a few days, and the reaction was changed from negative to positive. Bass then thought that by repeatedly testing a large number of infants and young children, latent tuberculous infection would often be revealed. The following procedure was carried out. Each child was subjected to a von Pirquet test, observed after twenty-four and again after forty-eight hours. A positive reaction was regarded as evidence of tuberculous infection. If the test was negative, a second von Pirquet test was performed, and a third, if necessary. If these tests were both negative, an intradermic injection of 0.1 c.c. of 1 : 1,000 of old tuberculin (0.1 mg.) was given. If this was negative after forty-eight hours, 0.1 c.c. of a 1 : 100 solution (1 mg.) was injected and repeated after forty-eight hours if the result was still negative. If after these six tests the child still failed to react, he was considered definitely negative.

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34. Sill, E. M.: The von Pirquet Reaction in Children, New York M. J. **107**:1018 (June 1) 1918.

35. Bass, M. H.: Cutaneous and Intercutaneous Tuberculin Tests in Infants and Children, Am. J. Dis. Child. **15**:313 (May) 1918.

In all, 1,165 tests were made on 206 children, varying in age from a few days to 6 years. Fifty-two children showed a positive reaction to some of the tests, or 25.2 per cent. If only one or two tests had been made, incorrect conclusions would have been drawn as to the number of children infected. It was interesting to note the steplike increase in the disease incidence, with increasing age. Out of fifty-one children under 6 months of age, four reacted positively, or 7.8 per cent. Of children in the second 6 months, 15.8 per cent. were positive; of those from 1 to 2 years old, 17.2 per cent. were positive; of those from 2 to 3 years old, 31.3 per cent. were positive and, finally, of the children between 5 and 6 years old, 78 per cent. were positive.

The author's results emphasize the fact that any series of examinations designed to ascertain the incidence of tuberculous infection must consist of more than a single test in each case. They show the great frequency of positive reactions in children.

Bass's report is timely, and emphasizes the importance of repeating the tuberculin test before the patient is pronounced free of tuberculosis.

J. P. Garrahan and L. Iraola [36] attempted to ascertain the frequency of latent tuberculosis in children. They examined 1,214 children from poor homes and two orphan asylums at Buenos Aires and found that 75 per cent. of the children, even though apparently healthy, responded positively to tests for tuberculosis. The proportion of positive reactions was 20 per cent. less among the children who had lived all or part of their life in the asylum than among the children who lived at home.

L. M. Spolverini [37] has made a study of latent tuberculosis in infants. He applied the tuberculin tests to 900 supposedly nontuberculous children under 1 year of age, and obtained a positive reaction in 63, or 7 per cent. Of the eight infants, 3 or 4 months old, he obtained reactions in only 0.80 per cent.; of twenty-two infants from 4 to 6 months old, 2.44 per cent. reacted, and of thirty-three infants from 5 to 12 months old, 3.66 per cent. reacted. The writer emphasizes again that contagion in the family is most likely to be the source of infection in infants, and that the first apparent localization of the disease is in the tracheobronchial glands.

A. Arnfinsen [38] tested 7,969 school children with the von Pirquet tuberculin test. He obtained a positive reaction in 37.8 per cent. of

36. Garrahan, J. P., and Iraola, L.: Latent Tuberculosis in Children, Rev. méd. del Uruguay **22**:459 (June) 1919.

37. Spolverini, L. M.: Latent Tuberculosis in Infants, Riv. di Clinica Pediat. **17**:169, 1919.

38. Arnfinsen, A.. Tuberculin Skin Tests in Children, Norsk Mag. f. Lægevidensk. **80**:508 (May) 1919.

the 6,978 children attending the public schools and in 33 per cent. of the 991 children attending private schools.

E. Friedman [39] performed the von Pirquet test on 464 children of Colorado. The desire to ascertain in what manner the climate of Colorado modifies tuberculous infection and tuberculous disease in children was responsible for the work. Nearly all the children tested were living under conditions comparable with those prevailing among the poorer working classes of the large cities. Most of the children were of tuberculous parents, many of whom had been inmates of sanatoriums, where they had had thoroughly inculcated rules of prophylaxis.

Of the 464 children tested, 39.8 per cent. reacted, and the number of positive reactions among the males was 2 per cent. greater than in the females. Of children less than 1 year of age, almost 12 per cent. reacted positively; from 10 to 14 years, 55 per cent. reacted positively, and from 14 to 18 years only 51 per cent. reacted positively. The highest number of positive reactions was noted from the eleventh to the thirteenth year. These figures do not show the uniform increase from year to year in the percentage of positive reactions which characterize the findings of other writers, and Friedman is at a loss adequately to account for this deviation.

The author found that of children not exposed to tuberculosis, less than one half as many react than of those exposed, but he did not find that children exposed to a closed case of tuberculosis reacted less frequently than those exposed to an open case. He was not able to corroborate the statement that tuberculous infection exercises a retarding influence on physical growth, or that there is a causal relation between measles and whooping cough and tuberculous infection.

Friedman draws the following conclusions from his study: The climate of Colorado exercises no striking influence in the prevention of tuberculous infection in children of tuberculous parentage. Among the nonexposed children, however, the frequency of infection is comparatively lower than obtains elsewhere. Active tuberculosis is conspicuous by its infrequency, and, when it occurs, it is exceedingly mild, with a tendency to prompt and complete healing. Among Colorado children fatalities to be ascribed to tuberculosis are so rare as to be negligible. This infreqnency of tuberculous infection in Colorado born children, the tendency to localization and to rapid recovery in the event of activity, and the low death rate all testify to the beneficial effects of the Colorado climate on the prevention and arrest of tuberculosis in childhood.

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39. Friedman, E.: The von Pirquet Test and Results of Its Use in Four Hundred and Sixty-Four Cases, Colorado Med. **16**:246 (Oct.) 1919.

G. H. Cattermole [40] made the tuberculin skin test on 419 children. Of 165 private patients, 42 per cent. gave a positive reaction and 58 per cent. gave a negative reaction; of 254 charity patients, 50 per cent. gave a positive reaction. Of the 254 charity patients, 167 were children treated at the University Dispensary, and eighty-seven came from an orphan home. Of the first group, 53 per cent. were negative, even though a large number of these children came from families where there were one or more cases of tuberculosis. In the second group, 57 per cent, gave a positive reaction. This was the highest percentage of positive reactions obtained in any group of children. At first, it seemed quite remarkable, as these children appeared to be well nourished and in better than average health, but the explanation lay in the fact that they came from homes in which one or both parents had died of tuberculosis.

In the children with positive reactions a healthy appearance is the rule. Ordinarily, they have but few signs in the chest, but when they contract any infectious disease the signs become apparent and persist for a longer time than in normal children.

The attempt to get a history from all children tested was successful in 202 cases. Of 101 children who gave a positive reaction, ninety-two had been exposed to tuberculous parents; sixty-one had been exposed to infection. This is a little more evidence that exposure in the home is the chief cause of tuberculous disease in children.

H. Heiman [41] has attempted to determine the value of the fixation reaction for the diagnosis of tuberculosis among infants and children. The blood serum of fifty patients, ranging in age from 6 months to 12 years was tested. Seventeen children were tuberculous, five were considered as being probably tuberculous or at least open to suspicion, and twenty-eight were nontuberculous. Among the tuberculous group were six cases of tuberculous meningitis, seven cases of pleural effusion, six cases of pulmonary tuberculosis, one of peritonitis, one of adenitis and one of tabes mesenterica. The complement fixation tests for tuberculosis on the serums of seventeen tuberculous infants and children with Miller's and Petroff's antigens gave one 4 plus, one 2 plus and two suspicious reactions. Complement fixation tests on the serums of five children, probably but not definitely tuberculous, were all negative with both antigens. Complement fixation tests on twenty-eight nontuberculous children gave three 4 plus and two suspicious reactions. In view of the very favorable reports given by others with the same anti-

40. Cattermole, G. H.: Tuberculosis in Children, Northwest Med. **15**:362, 1917.

41. Heiman, H.: The Complement Fixation Test for Tuberculosis in Infancy and Childhood, Arch. Pediat. **36**:32 (Jan.) 1919.

gens in general groups of individuals, Heiman would like to learn the experiences of others in this field, before discarding the test for children.

A. Reis [42] has attempted to verify the findings of Bath that there is a direct relationship between the tuberculin reaction and the chest circumference and height of the child. Of 125 children from 3 to 10 years of age, eighty-four boys and forty-one girls, thirty-six gave a positive reaction. Of these, twenty-one were below the normal height, fourteen had a smaller and five had a greater chest circumference than normal. Reis concludes from his study that there is no relation between the von Pirquet tuberculin test and the height, but children who have chest circumferences below normal show a larger percentage of positive reactions than do those who measure up to normal.

Mary E. Lapham [43] has made a study of the roentgen-ray findings of the chests of 150 children in order to ascertain how often are found in the lungs structural changes resembling those found in the chests of tuberculous children. Four groups of structural changes were found: (1) moderate enlargement of the bronchial glands, with little or no infiltration of the lungs; (2) enlargement of the bronchial glands, with beginning infiltration of the lungs; (3) considerable enlargement of the glands with marked infiltration of the lungs; (4) changes typical of tuberculous conditions. Although none of the children had the clinical features of tuberculosis, many had changes typical of the disease, which could be detected by the roentgen-ray. Lapham concludes from these roentgen-ray studies that tuberculous processes originate in the bronchial glands and extend from them into the lungs; that enlarged bronchial glands and thready infiltration of the lungs occur commonly in children, but that their presence need not be associated with the disease called tuberculosis. They occur whenever secondary complications take place in the terminal areas of the bronchi. Structural changes in the lungs, detected either on roentgenograms or by physical signs or by both, do not warrant the diagnosis of tuberculosis as a disease. Lapham feels that a more intensive study of changes in the nerves and the ductless glands, due to tuberculous toxemia, is necessary before all deviations from health, which may represent a tuberculous etiology, can be recognized. She believes that many of the functional diseases of children are manifestations of an occult tuberculosis.

The value of the roentgen-rays as an aid to diagnosis must not be underestimated, but, as Lapham points out, there are other conditions which cause pulmonary shadows very similar to those caused by tuberculosis. Especially since the epidemic of influenza, 1918-1919, one

42. Reis, A.: Relation Between Height and Chest Measure and Tuberculin Reaction, Arch. de méd. d. enf. **21**:26 (Jan.) 1918.

43. Lapham, M. E.: Tuberculosis and Roentgen-Ray Plates, New York M. J. **107**:294 (Feb. 16) 1918.

finds shadows in roentgenograms which undoubtedly a few years ago would have been attributed to tuberculosis, but which are due to the influenza capillary bronchitis and bronchopneumonia and the resulting enlarged bronchial glands. Moreover, if too much reliance is placed on the roentgenograms there is a tendency to make a less painstaking physical examination and to forget many of the signs which aided clinicians to arrive at a diagnosis long before the advent of the roentgen-ray.

A. A. R. Green [44] compared the physical signs in forty cases of early suspected pulmonary tuberculosis with results found on examination by the fluorescent screen. He says the points to which attention should be paid in screening a chest in a suspected case of tuberculosis are as follows: 1. The condition of the apices as regards translucency and the degree of illumination on coughing. 2. The condition of the structures at the hilus, whether, if the hilus is wider than usual, the enlargement is uniform in type or netlike, whether there are calcified areas or fibrous processes extending peripherally into the lung tissue. 3. Haziness, circumscribed or diffuse, of the lung tissue. 4. the movement of the diaphragm.

Green thinks, when physical findings are at variance with the roentgen-ray findings, greater stress should be laid on the second point. His reasons for this are that the sound-conducting power and resonating property of muscle vary with its tone, but the permeability of muscle to the roentgen-ray is usually the same. It is true, too, that deep seated areas of deficient aeration may be out of reach of percussion, yet may show almost as clearly on roentgen-day examination as though they were on the surface.

L. Ribadeau-Dumas [45] gives a detailed description of the roentgen-ray findings in pulmonary and glandular tuberculosis among infants and young children, and lays stress on their value as a diagnostic aid. Roentgenograms of pulmonary glandular tuberculosis in infants show at the base of the lung, a little above the diaphragm, a triangular shadow with its base toward the axilla. The apex meets the apex of another shadow, whose base corresponds to the mediastinum. The first shadow represents the perituberculous congestion around the initial lesion, the second, the enlarged bronchial glands. The evolution of the disease can be followed with the roentgen-ray. In cases which progress favorably, the shadow of the perituberculous congestion disappears, but that of the tracheobranchial glands becomes more prominent. Still later,

44. Green, A. A. R.: Comparison of Physical Signs in Early or Suspected Pulmonary Tuberculosis with Results Found on Examination by the Fluorescent Screen, Brit. J. Tuberc. **12**:153 (Oct.) 1918.

45. Ribadeau-Dumas, L.: Roentgenology of Tuberculosis, Progrès méd. **34**:372 (Sept. 20) 1919.

when the shadow of the perituberculous congestion disappears entirely, a small opaque area, which represents the remains of the primary focus, can be seen near the center of the posterior border of the axilla. In unfavorable cases the roentgenogram show extension of the primary focus. Sometimes the primary focus is never found, and only shadows of enlarged bronchial and pulmonary glands appear on the plate. Shadows of bronchial glands are seen most frequently on the right side. They represent either extrapulmonary or intrapulmonary glands. The extrapulmonary glands are those surrounding the trachea and bronchi, and the shadows extend from the base of the heart to the superior orifice of the thorax. Intrapulmonary glands cause shadows which sometimes are difficult to distinguish from hilus gland shadows. At other times they cause shadows radiating toward the periphery of the lung, in the region of the bronchi and blood vessels.

A. Weil [46] says that the roentgen-rays should be used as an aid to diagnosis, not to establish a diagnosis. Important findings in the roentgenograms are: 1. Diminished transparency of the pulmonary apices. 2. Diffuse shadows in the region of the hilus. 3. Diminution of the excursion of the diaphragm on the affected side. 4. Lessened expansion of the thoracic wall, with sinking in of the ribs. 5. Lengthening out of the cardiac shadow.

P. W. Roberts [47] says that until recently the diagnosis of tuberculous joint disease was considered one of the simplest and most definite problems with which the orthopedic surgeon is confronted, but from his studies on congenital syphilis, pursued during the last two years, he thinks that this simplicity is merely apparent. His records of more than 100 cases collected during the last two years give proof that a similar symptomatology may be produced by tuberculosis and inherited syphilis, and the roentgenographic findings show no differentiating characteristics. The Wassermann reaction in the present state of development is frequently negative in the presence of a direct family history of syphilis and there is no reliable laboratory test for tuberculosis, but fortunately nearly every case of joint disease due to inherited syphilis will, on exhaustive research, reveal some remaining imprint of the previous activity of the disease. The author lays special emphasis on the examination of the teeth in the differentiation of syphilitic from tuberculous joint disease. The deciduous teeth, the first permanent molars and incisors, are the ones which are most likely to show evidence of syphilis. The two conditions which the author finds most characteristic of syphilis are the widely spaced incisors and the "humpy

46. Weil, E. A.: Roentgen-Ray Signs of Tuberculosis, Paris méd. **8**:190 (March 2) 1918.

47. Roberts, P. W.: Syphilitic Joint Disease Simulating Tuberculosis, J. A. M. A. **70**:372 (Feb. 9) 1918.

molars," a term he uses, when there are alterations in the normally smooth lingual surface of the crown. In addition, he describes other alterations, early decay of both the first and permanent sets, the retention of deciduous units long after the period when they are usually displaced, and microdentition, as associated with congenital syphilis.

PROGNOSIS

L. Ribadeau-Dumas [48] and H. Béclére report a case of recovery from pulmonary tuberculosis in a child 13 months old. At the age of 9 months the child was put in the care of a nurse with an active tuberculosis. At 13 months he began to loose weight and had a slight elevation of temperature. A month later examination showed absolute dulness with disappearaace of the normal vesicular breath sounds over the entire right side. The temperature elevation persisted for three weeks, when the area of dulness gradually diminshed. The infant was very emaciated, had attacks of coughing, which resembled whooping cough, and a positive tuberculin test. There were also physical signs of enlarged tracheobronchial glands.

The child recovered, and the author had an opportunity to examine him six years later. There were no abnormal signs on examination of the chest, but the roentgen ray showed in the fifth interspace in the right posterior axillary region a small triangular shadow, probably a calcified tubercle, as well as small round shadows at the hilus (bronchial glands).

A. Grismondi [49] discusses the papulonecrotic tuberculid and its grave prognostic significance. The literature cited the outcome in each case when this form of tuberculid is present. Papulonecrotic tuberculids occur on the back, buttocks and limbs. In none of Grismondi's cases did an attack of measles precede their appearance, though this has been reported by other observers. In one case, however, a post-scarlatinal broncho-pneumonia accompanied a widespread eruption of tuberculids. Grismondi calls attention to the fact that papulonecrotic tuberculids may be the only sign of tuberculosis, and that their presence may clear up many a puzzling diagnosis.

PROPHYLAXIS

A. F. Hess [50] points out that statistics of infant mortality showing the ratio of deaths from tuberculosis as compared to deaths from other diseases do not give a true conception of the importance of tuberculosis at this period of life. To arrive at a better understanding of this

48. Ribadeaux-Dumas, L., Béclére, H.: The Initial Lesion in Pulmonary Tuberculosis in Infants, Bull. et mém. Soc. méd. d. hôp. **43**:561, 1919.

49. Grismondi, A.: Tuberculids in Diagnosis and Prognosis of Tuberculosis in Children, Pediatria **26**:635, 1918.

50. Hess, A. F.: The Significance of Tuberculosis in Infants and Children and Measures for Their Protection, J. A. M. A. **72**:83 (Jan. 11) 1919.

problem, figures must be prepared which show the absolute number of deaths annually. It is seen that in no one year do the deaths from tuberculosis exceed those of deaths occurring during the first year of life. This great loss is due, in part, to the peculiar susceptibility of infants, in part, to the fact that they receive an exceptionally large amount of the infective agent. Bovine infection plays a part that is minor but not negligible. In view of the not inconsiderable mortality that has been shown to be occasioned by the bovine type of bacillus, it would be well if cultural studies of tuberculosis in infants should be carried out from time to time, in order to gain an insight into the degree to which milk employed for infant feeding is contaminated by virulent tubercle bacilli.

Following the period of infancy, there is a quiescent phase in the mortality from tuberculosis, throughout which the deaths decrease year by year, although the number of infections, as shown by the tuberculin reaction, steadily increases. These infections are due probably to latent carriers, to individuals who from time to time shed tubercle bacilli. A daily examination of the sputum of a series of individuals, carried out carefully for a period of months, might well be undertaken in order to test the validity of this hypothesis.

There is a second active phase in connection with the mortality from tuberculosis. This is commonly associated with the third quinquennium of life. If the deaths are studied year by year, rather than by quinquenniums, it is found that this period begins at 13 years of age and is not coincident in females and in males. Among the former it sets in rather sharply at the age of 12, whereas among the boys the flare up does not manifest itself until the age of 16. This distinction was found to hold good for several large cities, and probably is governed by the onset of puberty. This uniform periodicity in the increase of deaths from tuberculosis, taking place in both girls and boys at a definite year of life, leads to the conclusion that the disease is due to an "autogenous reinfection" from some latent focus, rather than a fresh infection from a tuberculous individual.

It would seem that the best way to begin to combat this high mortality from tuberculosis would be to establish preventoriums for infants in connection with the large cities. These institutions should be situated in the country about two hours' train ride from the city. They should be built with many balconies, so as to provide for an outdoor life, and should be conducted by children's specialists according to the most approved methods. One or more wetnurses should be provided. The infants should be kept at the institution for at least a year. This central preventorium for infants could well serve as the administrative center for a system of boarding out infants in the farmhouses of the

surrounding country, under supervision of the physician and nurses of the institution; babies who were not thriving could be returned to the precincts of the preventorium for special care and treatment. There is good reason to believe that by this means thousands of babies in the large cities, who will be infected by their parents, could be saved and grow to be healthy. This question is particularly opportune and urgent at the present time, when many soldiers will return to their homes having communicable tuberculosis. The problem of the protection of older children against tuberculosis may likewise be said not to have been considered seriously in the United States.

One measure which the author suggests to guard against the tuberculosis of maturity is the establishment throughout the country of preventoriums similar to that at Farmingdale, N. J. To such institutions should be sent children who are poorly developed and undernourished and who give a positive tuberculin reaction. If it is true that infection at this period of life emanates from a focus within the body, then malnutrition should play a dominant rôle in the selection of the cases. These children should be kept in the country for from three to six months.

From the study of the mortality statistics, Hess concludes that it is inadvisable to make the age of admission from 4 to 14 years, as is done in France, since the high mortality does not begin until 12 years in girls and 16 years in boys. In view of the great need for preventoriums it would seem wise only to accept girls aged from 10 to 16 years and boys aged from 14 to 18 years, in other words, two years before the onset of the high mortality. With this change in the age of admission comes the advisability of providing separate preventoriums for boys and for girls. Finally, it seems possible that some affiliation might be made with agricultural schools, whereby these children could be referred to these institutions and trained in a permanent outdoor occupation.

Louis I. Harris [51] describes the measures carried out by the New York Department of Health to combat tuberculosis among children. The inspection and control of the milk supply by the bureau of foods and drugs from its source to its ultimate point of distribution is not only a preventive of gastro-intestinal diseases, septic sore throats and other infectious diseases, but also of tuberculosis. Pasteurization of milk, now mandatory in New York City, is preventive of tuberculosis among children.

The baby welfare stations, maintained by the department of health for the dietetic and hygienic guidance and supervision of infants, are also of value in the prevention of tuberculosis.

51. Harris, L. I.: What Are We Doing to Prevent Tuberculosis Among Children? Am. J. Pub. Health **8**:131 (Feb.) 1918.

Parents are taught to give special attention to proper treatment and aftercare for those who suffer from measles and whooping cough, diseases which are notoriously predisposing causes of tuberculosis.

The work of the bureau of child hygiene, which has as one of its chief functions the periodic medical examination of school children, may justly be regarded as the most effective weapon with which the department of health wages its attack against tuberculosis among children. The bureau exercises a constant supervision over 2,000 children. who have been exposed to tuberculosis and whose physical condition makes them susceptible to the disease. The bureau examines children between 14 and 16 years of age, who apply for working papers. This is a means of sifting out those who appear to be susceptible to tuberculosis, as well as those who give evidence of an already existing infection.

The examination of public school teachers in whom tuberculosis is suspected has resulted in the exclusion of a number of actively tuberculous teachers. School lunches are distributed free to ill nourished children. This is valuable chiefly as a palliative measure.

The twenty tuberculosis clinics of the department of health provide a means for the diagnosis, treatment and instruction of children exposed to tuberculosis, and of those susceptible or giving evidence of the disease. The department of health has equipped two ferry boats to care for adults who are unwilling or unable, or who are delayed in entering hospitals, and for exposed and infected children. It has provided for the care in special tuberculosis hospitals of 350 active cases of tuberculosis in children.

Harris thinks the segregation and forcible detention in Riverside Hospital of about 200 patients, who would not observe proper care in the home, has been most helpful, and that such detention deserves more general adoption. It would close one way of transmitting tuberculosis to children and others.

W. D. Beadie [52] outlines the following as preventive of tuberculosis among children: Prenatal care should be given expectant mothers. Children born of an actively tuberculous mother should be removed to a safe environment immediately after birth, and especially should an actively tuberculous mother be compelled to refrain from nursing her infant. Maids in a household, and those having the care of young children generally, should be examined for tuberculosis. Bovine tubercle bacilli have been found in pulmonary tuberculosis and in disease of the glands and joints. The laws requiring cattle to be tested should be more stringently enforced. Since pasteurized milk has been used more

52. Beadie, W. D.: Prevention of Tuberculosis in Children, Minnesota Med. **2**:181 (May) 1919.

generally, there has been a marked reduction of this form of infection in children. More work should be done along the lines of school inspection. Education of older children by physicians and health officers would aid greatly in the prevention of tuberculosis.

E. Friedman [53] reviews the literature on the frequency, portal of entry, pathology, symptoms, diagnosis and prophylaxis of tuberculosis in infancy and childhood. From his study, he says it is evident that the child must figure prominently in every movement calculated to check the progress of tuberculosis. The fundamental principle in prophylaxis consists in removing a child from a tuberculous environment, particularly during the first three or four years of life. It is at this period that the body defenses are wholly inadequate to cope with tuberculous infection and the toll exacted by death is appalling.

Armand-DeLille [54] describes the methods, particularly those of the "Oeuvre Grancher," instigated in France to prevent tuberculosis in children. This institution finds homes in the country where children of tuberculous parents can be placed. Children from 3 to 15 years of age are accepted, but no child who is ill or has signs of latent tuberculosis is eligible. The children are first taken to the central office of the organization, where they are fitted out with clothes and operated on for adenoid vegetations if necessary. They are then taken to their destination. A physician makes daily visits, and once a year officers of the society make rounds of inspection. The results have been marvelous; none of the children have become tuberculous. At the age of 13, if the parents are living and desire the children, they are returned to them. Occasionally a child is recalled by the death of the tuberculous parent. At first the society had only twenty-seven patients; before the war the number had increased to 810; at present there are 400 in residence.

C. Ferreira [55] inaugurated in Brazil a movement for the care of children of tuberculous parents similar to that of Grancher in France. The work was begun in 1908, and in 1916 fifty-eight children, forty-two girls, aged from 2 to 16 years, and sixteen boys, aged 2 to 10 years, were being cared for. At present, the organization has a large sanitarium at Brazanca and is planning two more sanitariums and two preventoriums.

C. Ferreira [56] finds that tuberculosis causes only 1.2 per thousand of the general mortality of the children of S. Paulo. In fourteen years (1902-1916) there had been recorded 186 deaths from pulmonary,

53. Friedman, E.: Tuberculosis in Infancy and Childhood, Colorado Med. **16**:8 (Jan.) 1919.

54. Armand-De Lille, P.: Campaign Against Infantile Tuberculosis in France, J. A. M. A. **71**:1 (July 6) 1918.

55. Ferreira, C.: Tuberculosis in Children, Ann. Paulistas d. Med. e. Cir. S. Paula **10**:97, 1919.

56. Ferreira, C.: Protection of Children of Tuberculous Parents, Brazil-med. **32**:153 (May 18) 1918.

eighty-three from abdominal tuberculosis, and forty-three from tuberculous meningitis, a total of 350 deaths from tuberculosis among children under 10 years of age. During the last few years a vigorous campaign against tuberculosis has still further reduced this mortality rate.

A. F. Hess [57] reports the results obtained in the preventorium at Farmingdale. During the two years of its existence, thirty-six infants, from 1 month to 1 year of age, were admitted. A number of these weighed less than seven pounds. In all but one instance the mother was tuberculous, and in all but seven instances did the mother's sputum contain tubercle bacilli, so evidently the mother was the most frequent source of infection. During the first few months, infants already infected, as shown by a positive tuberculin reaction, were admitted to the preventorium, but it was found impossible to prevent the rapid development of tuberculosis in them, so later only those giving a negative reaction were received. In all twenty-eight infants, with a negative tuberculin reaction, have been cared for.

Of these, five died, two from diphtheria, one from enteritis, two from marasmus. Hess does not regard these results as being discouraging, and does not hesitate to give the opinion that it is quite possible to save infants of tuberculous mothers. Moreover, he feels it a duty to provide for these infants. At present, they are abandoned to their fate and are only individuals left out of consideration in the tuberculosis propaganda being spread so rapidly through the country.

P. Nobécourt and J. Paraf [58] call attention to the hospital wards as the source of tuberculous infection in infants, reporting two instances of infants evidently contracting the disease in a hospital creche. The first patient never left the hospital as the mother remained there as a nurse. The child developed well until 11 months old, when she contracted whooping cough; then there was loss of appetite, diarrhea and fever, with dullness, bronchial breathing, and crepitant râles at the base of both lungs. The diagnosis of tuberculosis was confirmed by the roentgenoscope and tuberculin reaction.

The second child developed well until the fifth month, when she showed evidences of a tuberculous bronchopneumonia. The child died two months later and necropsy confirmed the diagnosis.

Hospital infection may be due to direct infection from a nurse or attendant or indirect infection from a contaminated ward. Two tuberculous infants had been in the ward shortly before the children referred to by the authors which emphasizes the importance of disinfection after removing a tuberculous patient.

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57. Hess, A. F.: Tuberculosis Preventorium for Infants, Am. Rev. Tuberc. **1**:669 (Jan.) 1918.

58. Nobécourt, P., and Paraf, J.: Tuberculosis of Infancy, Arch. de méd. d. enf. **22**:355 (July) 1919.

R. M. Smith [59] discusses the danger to children from tuberculous cattle. He believes that, although bovine tuberculosis represents only a small part of the disease in children, it is very important and can be prevented. It has been estimated that 25 per cent. of all cases of tuberculosis in children under 5 years of age are of bovine origin, and that in New York City from 6 to 10 per cent. of the children who die of tuberculosis each year in the hospitals die of bovine tuberculosis. Smith investigated a series of sixty-seven cases of bone tuberculosis in children, and found that 70 per cent. were of bovine origin. Of the cases in children under 1 year in this group, all were of bovine origin, and under 4 years, 78 per cent. were of bovine origin. Smith says that the real hope of eliminating bovine tuberculosis rests in prevention, not treatment. As infection comes exclusively from milk or milk products, cream, butter, ice cream and cheese, all milk must be pasteurized, unless it is positively known to come from a herd free from tuberculosis. But milk must be pasteurized properly or false standards are established, and physicians should insist on some sort of inspection and supervision of pasteurizing plants.

C. M. Hilliard [60] found that at least 10 per cent. of the dairy cattle supplying milk to infants, children and adults are infected with tuberculosis. The problem of prophylaxis resolves itself into the elimination of tuberculous cattle or the pasteurization of milk. The author believes that one of the most important functions of the antituberculosis league is to bring about compulsory pasteurization of milk.

E. Maragliano [61] brings to date his report on the vaccination of children and adults against tuberculosis, which he has been advocating for twenty-five years. The method is to introduce dead tubercle bacilli subcutaneously, and he believes that from this focus will be produced antigens which will pass into the circulation and slowly and progressively induce the production of immunizing bodies. He has proved by clinical and biologic tests that antibodies are actually produced. The vaccination was applied from 1907 to 1914 to 3,702 members of families with tuberculosis. The history of 1,893 has been obtained up to 1915. Of this number, 1,819 were living, sixty-three had died from other than tuberculous processes, and only eleven had died from tuberculosis. All persons vaccinated in 1907 were found to be in good health. In twenty-six persons vaccinated earliest, persistence of immune bodies and antibodies was demonstrated. Maragliano believes

59. Smith, R. M.: Danger of Children from Tuberculosis in Cattle, Boston M. & S. J. **177**:657 (Nov. 8) 1917.

60. Hilliard, C. M.: Tuberculosis in Dairy Cattle, Boston M. & S. J. **177**: 654 (Nov. 8) 1917.

61. Maragliano, E.: Vaccination Against Tuberculosis, Riforma med. **35**: 542 (July 5) 1919.

that the only efficient means of prophylaxis against tuberculosis is to acquire a specific resistance against the bacillus, and he feels that his work has opened a practical means to this end.

W. C. Rivers [62] believes that school attendance is actually responsible for the greater part of juvenile phthisis, and from statistics based on medical literature, and his own experiences, he draws the conclusion that there is much more phthisis among children of school age than among those under this age. He believes that school attendance predisposes to tuberculosis by lowering the general health through the indoor confinement and restraint.

TREATMENT

Meyer-Solis Cohen [63] has made a comparative study of the therapeutic effects of various forms of tuberculins, vaccines and serums in pulmonary tuberculosis in children. He reports the results in fifty-one children.

Ten boys and nine girls, from 2½ to 15 years of age, were given Koch's old tuberculin. Seventeen were in the incipient stage of the disease, one was in the moderately advanced stage, and one in the far advanced stage. The initial dose varied from one-billionth to one-millionth of a milligram. The dose was increased in thirteen cases but held in six. It was not decreased in any. The largest dose given was one five thousandth of a milligram. The interval between doses was from three to five days. A favorable reaction followed one or more of the doses in thirteen children; an unfavorable one resulted in sixteen children. In one child no reaction was observed. Benefit from the tuberculin treatment was distinctly noted in thirteen of the children. Six were not benefited by it. None were harmed.

Bacillen emulsion was given to fourteen children. Twelve were in the incipient and two in the moderately advanced stage. The initial dose varied from one-hundred millionth to one millionth of a milligram. The initial dose was increased in every case, the highest being one five hundred thousandth of a milligram. The interval between doses was from three to five days. The tuberculin seemed to be beneficial to nine patients, of no benefit to five and harmful to none.

Tuberculin Ruckstand was administered to thirteen children Seven were in the incipient stage, four in the moderately advanced stage and one in the far advanced stage. The initial dose varied from one-hun-

62. Rivers, W. C.: Juvenile Consumption and School Attendance, Brit. J. Child. Dis. **15**:6 (Jan.-March) 1918.

63. Solis-Cohen, M.: Comparative Study of Therapeutic Effects of Various Forms of Tuberculins, Vaccines and Serums in Pulmonary Tuberculosis in Children, Arch. Pediat. **35**:11 (Jan.) 1918.

dred-millionth to one-millionth of a milligram. Twelve patients seemed distinctly benefited during the treatment, one was not benefited, none were harmed.

Six boys and four girls were given bouillon filtrate by mouth. Eight were in the first stage of the disease, one was in the second, and one was in the third stage. The initial dose was from one-hundred-millionth to one millionth of a milligram. Six patients appeared to be distinctly benefited; four seemed unaffected; none seemed harmed.

Eleven children were given two different forms of tuberculin at the same time, seven were benefited, and in four there was no effect. Six children, all in the first stage of the disease, were given Sherman's nonvirulent tubercle bacilli vaccine. The effect was beneficial to one patient, harmful to one, and negative to four.

Seven children were given both Sherman's nonvirulent tubercle bacilli vaccine and Sherman's catarrhal combined vaccine. Six were in the first stage and one was in the second stage of the disease. The general results seemed beneficial in two cases, harmful in one and negative in four.

Six children were given Bruschettini's serum vaccine. One patient seemed benefited, one seemed worse, four seemed unchanged during the treatment. Seventeen of the patients were given at different times several forms of tuberculin, vaccines and serums. None of the combinations of different forms of tuberculin seemed to be of much value.

Cohen thinks it is impossible to draw positive conclusions from the small number of cases studied. But it would seem, that the kind of tuberculin, vaccine and serum used did have some influence on the results obtained. The tuberculins gave better results than the vaccines and serums. Tuberculin Ruckstand to a marked degree and old tuberculin to a slighter degree, properly administered, offer hope of benefiting children with pulmonary tuberculosis. Bacillen emulsion, bouillon filtrate, Bruschettini's serum vaccine and the simultaneous employment of Sherman's non-virulent tubercle bacilli vaccine with Sherman's catarrhal combined vaccine, do not give results to warrant their being rcommended. Cohen advises, however, that when one form of tuberculin fails to be of benefit another form may be.

E. Lackner [64] has had a favorable experience with the tuberculin treatment of tuberculosis in children, but he believes much more work must be done in this direction. He advises an initial dose of 1/1,000 mg. of old tuberculin, gradually increased to 1 c c, which he reaches after from nineteen to twenty injections, given at five-day intervals. As a rule, he observes the patient's temperature for three days before

64. Lackner, E.: Tuberculin Treatment of Tuberculosis in Children, Illinois M. J. **36**:76 (Aug.) 1919.

commencing treatment, taking two hourly temperatures during the day and four hourly at night. After the injection of tuberculin, the same procedure is followed. A rise of from 1 to 3 degrees over anything recorded previous to the injection is regarded as a reaction. After the tolerance of the patient is determined, treatment can be continued without a reaction. Lackner is fully convinced that the dose of tuberculin must not be so large as to cause a reaction. It must always be well diluted, in order to prevent focal reactions and reactions at the point of injection, and should be given at five-day intervals. For the length of treatment, Lackner quotes Koch, who gives an average of twelve weeks for pulmonary and bronchial gland tuberculosis; thirty-five weeks for tuberculosis of the pharynx and larynx, seven weeks for tuberculosis of the pleura, ten weeks for tuberculosis of the peritoneum, eleven weeks for tuberculosis of the bones.

Lackner believes that latent forms of tuberculosis are positively helped with tuberculin and the forms occurring between 5 and 10 years are more easily affected than those in the earlier and later periods. Miliary tuberculosis, severe affection of both lungs, and degeneration of the parenchymatous organs are contraindications to the use of tuberculin.

De los Terreros [65] has treated twelve children with various forms of tuberculosis with Spengler's immune bodies. He gave the treatment a thorough trial for four years. The results were good in six children; fair in three and in three the disease continued a progressive course. Two of the children died. The outcome was better the earlier the stage and the older the child.

R. C. Newton [66] reports seven cases of tuberculosis in children treated with tuberculin, which he thinks illustrate several important truths in tuberculin treatment, namely: the frequent unexpected benefit of small doses of the drug; the great importance of having the patients under treatment at the earliest possible moment, the necessity of at times changing the form and dosage of the tuberculin; the desirability of using vaccines and different agents, in conjunction with the tuberculin, the importance of every available hygienic measure; the treatment of children in the pretuberculous stage.

The author's cases include four cases of tuberculous peritonitis and three of pulmonary tuberculosis. Of the four cases of peritonitis, three patients were brought under treatment within a few weeks of the appearance of the ascites and the improvement was prompt and gratifying. The fourth patient took much longer to recover. The

65. de los Terreros, C. S.: Spengler's Immune Bodies in the Treatment of Tuberculosis in Children, Arch. españ. de pediat. **3**:263 (May) 1919.

66. Newton, R. C.: Tuberculin Therapy in Children, Med. Rec. **96**:459 (Sept. 13) 1919.

three patients with pulmonary tuberculosis also improved under treatment. The author mentions excellent results in three other patients with pulmonary tuberculosis, who came under his care within a few months of the beginning of active lesions. He emphasizes the importance of early treatment, in order to obtain permanent benefit.

J. P. Garrahan [67] reports his experience with the intradermic injections of tuberculin for diagnosis and treatment in 653 children from 2 to 16 years old. As a diagnostic test he found it more reliable than the von Pirquet test, especially in instances of latent tuberculosis. When suspicious of hypersensitiveness, he advises giving a dose of 0.00001 gm., instead of the usual 0.0001 gm. A positive reaction in an infant is an almost unmistakable sign of active tuberculosis and a repeated negative reaction in older children certainly excludes tuberculosis. According to Garrahan's opinion, treatment with intradermal injections of tuberculin is absolutely harmless, and there does not seem to be any contraindication to its use. He does not, however, share Combe and Jeanneret's optimism as to its efficacy. He advises it as an adjuvant or when climatic and hygienic treatment is not available.

R. Hertz,[68] who has charge of a sea coast sanatorium for children in Denmark, has given heliotheraphy a very prominent place in the treatment of tuberculous disease of the bones and joints. The sun baths are taken for about six hours a day and the children recline or play or stroll about in the open air unclothed. In stormy weather they remain indoors in a large hall built like a green house. The reflection from the water of the bay enhances the effect of the sunlight. The children seem to bear low temperatures better when they are well tanned.

Hertz's results nearly equal those obtained by Rollier at Leysin and he asserts that the cure has been permanent in a much larger number of cases than with the best surgical procedures.

R. C. Newton and S. A. Twinch [69] compare the results in fifty cases of tuberculosis, mainly osseous and glandular, treated with tuberculin. With a few exceptions, these patients were children, inmates of a home for crippled children, and patients in a hospital clinic. Without tuberculin, 16 recovered, or 32 per cent.; 25 died, or 50 per cent.; 9 are still wearing braces and are under treatment, or 18 per cent. With tuberculin, 33 recovered, or 66 per cent.; 5 were much improved, or

67. Garrahan, J. P.: Treatment of Tuberculosis in Children, Prensa med. Argentina **6**:35, 1919.

68. Hertz, R.: Conservative Treatment of Surgical Tuberculosis, Ugesk. f. Læger **80**:196, 1918.

69. Newton, R. C., and Twinch, S. A.: Fifty Cases of Tuberculosis, Mainly Osseous and Glandular, Treated with Tuberculin, Contrasted with Fifty Cases Treated without Tuberculin, Med. Rec. **94**:407 (Sept. 7) 1918.

10 per cent.; 6 died, or 12 per cent.; 5 are still under treatment, or 10 per cent.; 1 discontinued treatment, or 2 per cent. Of the 16 patients cured without tuberculin the average period of treatment for each case was five years and three months. Of the 33 cures with tuberculin, the average period of treatment for each case was eleven and one-half months. Of the 25 deaths without tuberculin the whole period of treatment was seventy-two years, eleven months, nine days, or three years and fourteen days for each patient. Of the 6 deaths among those treated with tuberculin the total period of treatment was four years, nine months and twenty-nine days, or an average of nine months and thirteen days for each patient. Of those treated without tuberculin, 9 are still under treatment. Of those treated with tuberculin, 5 are still under treatment.

E. Stolkind [70] discusses the treatment of pulmonary tuberculosis by means of artificial pneumothorax. He says the ideal case is one of advanced pulmonary tuberculosis in one lung, with the other lung at least, clinically normal. The treatment, however, is still indicated, if the disease in the other lung is inactive, chronic or quiescent. Artificial pneumothorax is useful also in acute unilateral pulmonary tuberculous pneumonia, in cases of pulmonary tuberculosis with pleural effusion, spontaneous pneumothorax, or seropyopneumothorax. In the latter instances, the fluid is gradually removed and the gas is introduced. The treatment is indicated also in cases of pulmonary abscess.

Pneumothorax treatment is contraindicated in cases of pulmonary tuberculosis, complicated by other severe diseases. Asthma, too, is a contraindication.

In the treatment with pneumothorax the same complications may occur in children as in adults. The most frequent complications are subcutaneous emphysema and pleural effusion. Subcutaneous emphysema occurs in the great majority of the cases. Usually, it is not extensive, is harmless, and disappears in a few days. More serious is the occurrence of deep emphysema, in which the gas burrows between the endothoracic fascia and parietal pleura or under the visceral pleura. Pleurisy with effusion is the other complication frequently observed. Suppurative effusion, pleural shock, pleural reflex and gas embolism are the most dangerous complications, but occur rarely.

Stolkind believes that the results of treatment with pneumothorax in subacute cases are very satisfactory. Generally, the temperature falls, the cough and sputum lessen, and the tubercle bacilli diminish or disappear. Children and adolescents usually gain weight. The author

70. Stolkind, E.: Treatment of Pulmonary Tuberculosis in Children with Artificial Pneumothorax, Brit. J. Child. Dis. **16**:18 (Jan.-March) 1919.

is of the opinion that pneumothorax treatment should be applied in every case of advanced pulmonary tuberculosis, not only in adults, but also in children.

E. Stolkind's advice as to the use of pneumothorax is dangerous, to say the least. Even if the disease is apparently limited to one lung, there is no guarantee that a small hidden focus in the other lung would not light up under the additional strain and cause either a widespread tuberculous bronchopneumonia or a miliary tuberculosis. If the disease in the other lung is chronic or quiescent, there is little doubt but that these catastrophes would occur.

M. H. Beiz [71] reports three cases of the ascitic form of tuberculous peritonitis treated with autotherapy. The first patient was a boy, 5 years old, who had been ill two months with nausea, vomiting, pain in the abdomen and fever. On entrance to the hospital there were signs of tuberculosis at the base of the right lung and a greatly enlarged abdomen. Fifty cubic centimeters of fluid were withdrawn, and 10 c c were injected into the muscles of the thigh. The injection was well tolerated and was followed by a polyuria. The abdomen gradually diminished in size and the ascites disappeared.

The second patient was a boy 11 years old, with a tuberculous ascites. Ten cubic centimeters of fluid was injected beneath the skin of the thigh, and one week later 20 c c were injected. Three other injections of 30 c c were administered at eight day intervals. Little improvement followed and resort was made to heliotherapy with good results.

The third patient was a boy, 10 years old. The first injection consisted of 15 c c. of fluid, which was followed in eight days by another injection. No results followed the injection.

In a fourth patient autotherapy did not give any better results, and from his experience the author does not advise its use. Moreover, he has shown that rest, compression and fresh air are usually sufficient to effect a cure of this form of tuberculous peritonitis.

Ramond and Francois [72] emphasize that tuberculosis is essentially curable, especially when it involves serous membranes. Their experience with injections of air during the last two years seems to suggest that this method will become valuable in the treatment of tuberculous meningitis. The procedure seems absolutely harmless with the following technic: The lumbar puncture is made with the patient in the reclining position, and about 40 c.c. of fluid is withdrawn. The air is

71. Beiz, M. H.: Autoserotherapy in Ascites of Tuberculous Peritonitis, Abstr., Arch. de méd. d. enf. **22**:99, 1919.

72. Ramond, F., and Francois: Treatment of Tuberculous Meningitis by Injections of Air Into the Spinal Canal, Bull. et. mém. Soc. méd d. hôp. **41**:1058, 1917.

drawn into a Roux syringe through a long, red hot platinum needle, which sterilizes and warms it. It is then injected slowly through the puncture needle, which has been left in place. The amount of air injected should not exceed one-half or two-thirds of the amount of fluid withdrawn. The injection should be repeated on five or six consecutive days, but no longer, as the nitrogen of the air is not readily absorbed. If oxygen is used, the injections can be kept up indefinitely. The authors' results with this form of treatment have been encouraging.

Bausa [73] deplores that he has never been able to save a child with tuberculous meningitis. He suggests several procedures in the treatment of this condition. The withdrawal of large amounts of spinal fluid, a method not dangerous, as cerebrospinal fluid forms rapidly; a canula might be left in the spinal canal to insure permanent drainage and to enable an antiseptic to be applied directly to the meninges. Heliotheraphy, local heat and antiserums might be used as adjuvants. Bausa believes that tuberculin might be of value during the prodromal stage.

William Aston [74] lays stress on the frequent examination of all delicate children under school age, and especially those with a tuberculous family history. He considers it a duty of utmost importance for all public authorities to see that sufficient attention is paid to the prevention and treatment of tuberculosis among children. A campaign should be carried out to establish convalescent camps in the country, open air schools and day sanatoriums for the early cases, and to provide proper institutional care for the reception of all children in whom the disease is definitely established.

V. Y. Bowditch [75] describes the children's pavilion at the Sharon Sanitarium. It is intended to be a combined sanatorium and school for debilitated children with suspected tuberculosis, and for children in the earliest stages of pulmonary and glandular tuberculosis. Children of parents of moderate incomes have preference in being admitted.

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73. Bausa, J. M.: Treatment of Tuberculous Meningitis, Am. Rev. Tuberc. **3**:51, 1919.

74. Aston, W.: The Care of Tuberculous Children, Brit. J. Tuberc. **13**:124 (July) 1919.

75. Bowditch, V. Y.: Children's Pavilion at the Sharon Sanitarium, Boston M. & S. J. **178**:553, 1918.